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HOWARD NAFFZIGER

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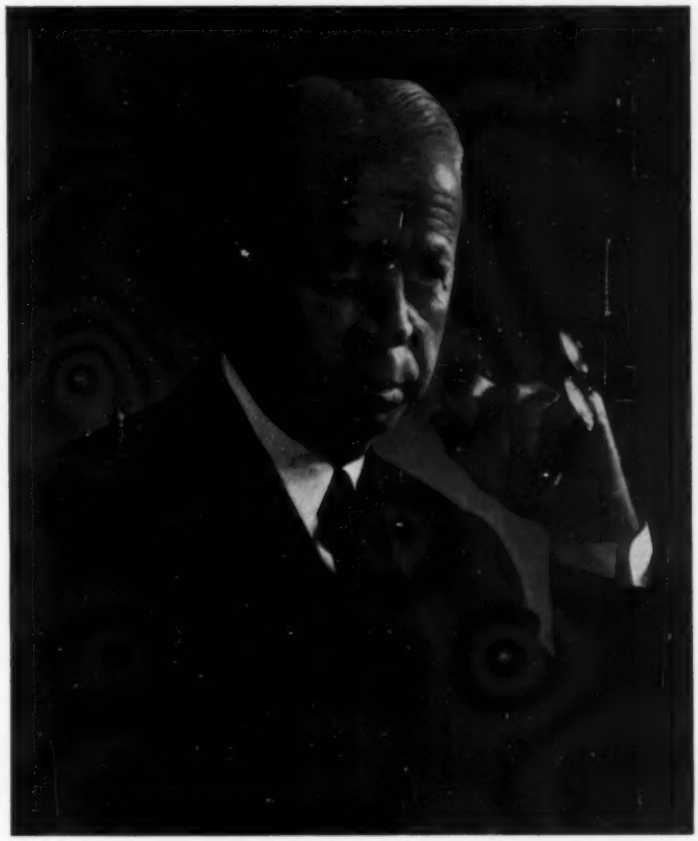
THE lives of some men are so colourfully patterned, patterned rather than designed because few of us can look back and claim that we have trodden paths presciently mapped out, that a sketch in black and white is an unsatisfying replica of the original. Dr. Howard Naffziger is a case in point. His years have flown by so fast that he will find himself on May 6 celebrating his seventieth birthday, some few years before he expected it. So much has happened during these years, a walk, a run, a gallop from the tin-mines near Nevada City to an honoured place as a Regent on the controlling board of the University of California, Berkeley, the first doctor to hold office in the inner Councils of that famous University.

In the meantime he has been Professor of Surgery, 1929–47, Professor of Neurological Surgery 1947–51,



Howard C. Naffziger

a Member, a Fellow, a Regent of the American College of Surgeons, and its relatively youthful President, 1938–39. California trained, he is 'a Halsted man' for he was Assistant Resident Surgeon at Johns Hopkins in 1911–12 and worked there with those two surgical giants, Halsted and Harvey Cushing, the second of whom would shortly leave for Harvard and Boston. Howard Naffziger preferred to

return to his native State, being a young giant himself, without following the Cushing star. In that he showed the individuality that has marked all his doings. He was too original to tie himself to another strong personality. In World War I he was a lieutenant-colonel, in World War II he wore the mufti which was outside and usually above the range of identification by rank, a wise choice since he was able to be Consulting Surgeon to the Navy and also to be a Member of the Medical Division of the National Research Council and Chairman of its Neurosurgical Committee, besides many extra-curricular duties. He is now Senior Civilian Consultant to the Surgeon General of the U.S. Army and went to Korea to report on battle conditions there and the management of casualties on the field.

Dr. Naffziger is, of course, a Member of many Societies and he has fifteen honorary memberships. He was made a Fellow of our own Royal College of Surgeons at its meeting in July, 1943. He has held office in the Editorial Boards of several famous journals and is a Director of *Surgery, Gynecology and Obstetrics* and the Franklin Martin Foundation. In surgery and in neurological surgery Dr. Naffziger has made many notable contributions and is affectionately regarded as one of the wise men of our profession

This abbreviated list of his attainments suffices to show the trust and admiration in which Dr. Naffziger is held by many bodies diverse in their activities. No man wins the enduring respect and confidence of his fellows without possessing the qualities which justify it. His is a strong personality, his fine critical judgement has not been blunted by the passage of the years. He has always been a most engaging man, his shrewdness tempered by so much good nature that he has never failed of a warm, even a boisterous, reception in any gatherings be they his own talented family or doctors, sportsmen, or statesmen. The part that he has played in sorting the wheat from the chaff in medicine, in penetrating pretension to its empty core will long be remembered. We greet him as a firm friend of this Country, as a colleague of whom we can be very proud, and tender him now our very sincere congratulations on a long record of achievement, a record happily not yet closed.

CARCINOMA OF THE HEAD OF THE PANCREAS

A REVIEW OF 49 PERSONAL CASES

By R. G. MOORE and J. D. YOUNGHUSBAND, Portsmouth

Carcinoma of the head of the pancreas, carcinoma of that part of the common bile-duct which lies within the head of the pancreas, and carcinoma of the ampulla of Vater may be grouped together as a single entity. The first two are usually indistinguishable except by the pathologist, and the third, although it possesses certain distinctive features, can seldom be distinguished from the other two before operation, and is usually treated by the same surgical procedures. Certain cases of carcinoma of the duodenum can be included in the entity for similar reasons.

This paper is an account of 49 consecutive cases of this entity treated by us in the Portsmouth Group Hospitals in the last five years, together with a consideration of the symptoms, diagnosis, and details of operative treatment.

INCIDENCE

The cases described are shown in *Table I*. We have been able to trace 13 cases treated by colleagues

Table I.—Summary of Cases

	Cases
Head of pancreas	33
Common bile-duct (within head of pancreas)	7
Ampulla	8
Duodenum (in close relation to ampulla)	1
Total	49

during the same period, so that the incidence in this group may be taken as 62 cases occurring in five years in a population which may be estimated at 350,000. A review of the literature has shown that the subject has been scantily recorded in this country and we have been unable to find a record of a large series of cases. In America, Brunschwig (1943), Whipple (1946), and Cattell and Pyrtek (1949) have recorded large series of cases treated in the last twenty years, and sufficient time has now elapsed for consideration of the long-term results of treatment. Unfortunately the results of radical treatment are not as good as had at first been hoped (Cattell and Pyrtek, 1949), and indeed, with modern advances, might reasonably be expected. However, Miller and Clagett (1951) record a case of a patient of 41 who had a carcinoma arising in the head of the pancreas with involved glands, and who was free from signs of recurrence five years after pancreatico-duodenal resection. This is apparently the first recorded five-year survival of its kind.

In this country there is probably no surgeon with a personal experience of this subject large enough as yet to be of statistical value. The first published successes were those of Maingot (1941) and Gordon-Taylor (1942, 1943). More recently d'Offay (1946) extensively reviewed the historical development of the radical operation and reported several successful cases of his own. Morley (1947) recorded 6 cases of carcinoma of the ampulla of Vater, Dodd (1949) recorded 2 cases, and Illingworth (1951) has reviewed the diagnosis and treatment, but, although the radical operation is probably being performed by many surgeons nowadays, reports in British literature are still conspicuous by their scarcity.

Rodney Smith (1953) has gone a long way towards filling the gap with his outstanding book *The Surgery of Pancreatic Neoplasms*. The difficulties of diagnosis and treatment are fully discussed in the light of published results, and he records 25 cases of this type of carcinoma, including 7 radical operations.

Another valuable contribution to the subject is the work of Falconer and Griffiths (1950), who have made a detailed study of the vascular supply of the pancreas and its frequent variations.

Our purpose in writing this paper is to record our experiences and difficulties, and the results of treatment, rather than to describe all aspects of the disease, and for this reason we have not thought it necessary to repeat much of the ground so thoroughly covered by these authors.

CLINICAL ASPECTS

Sex Incidence.—In this series there were 21 males and 28 females. This contrasts with the generally accepted ratio of males/females of 2–4 to 1 (Brunschwig, 1942) or 3–4 to 1 (Rodney Smith, 1953).

Age Incidence.—This is given in *Table II*. It will be seen that four-fifths of the cases were over 60 years of age, and one-third were over 70.

Table II.—Age Incidence

	Cases
30/40	2
40/50	2
50/60	6
60/70	22
70/80	14
80/90	3

Symptoms.—Jaundice is the outstanding symptom of this disease. It was present in every case in this series. Only two cases were referred for symptoms other than jaundice, one of whom complained of right-sided abdominal pain for five months, the other of backache for three months. Both these patients developed jaundice just after they were first seen by one of us. Fourteen patients had pre-icteric symptoms of three months' duration or more, consisting of anorexia, abdominal pain, backache, loss of weight, constipation, or diarrhœa, in some cases due to or coincident with intercurrent disease such as chronic cholecystitis. In the remainder, jaundice appeared within a week or two of the onset of illness, which often took the form of a mild digestive disturbance, or a sudden chill. Several patients noticed dark-coloured urine and pale stools as their first symptom, followed in a few days by clinical jaundice.

None of these pre-icteric symptoms can be said to be specific to carcinoma of the pancreas, but when a patient gives a short history of one or more of them this diagnosis must be considered. Cattell (1945) has stated that loss of weight, asthenia, and anorexia are early features in carcinoma of the head of the pancreas and usually precede the jaundice, but are less marked and usually follow the jaundice in ampullary carcinoma. We can confirm Cattell's findings in this series.

Jaundice.—It is fortunate, therefore, that the vast majority of these tumours obstruct the bile-duct at a relatively early stage. The jaundice which results has several important features. Commonly it is steadily progressive and may increase in depth slowly or surprisingly rapidly. In some cases, however, it is intermittent, and the colour of the stools and urine may vary from time to time. In a few cases, as Illingworth (1951) describes, the jaundice may disappear completely after a week or two, and this is a dangerous feature if it is allowed to deceive the medical attendant into concluding that a carcinoma cannot be present.

Pain.—Older text-books state that pain is absent, but modern writers agree that it is a prominent symptom. Brunschwig (1942) states that pain is a major complaint and may be the initial symptom in carcinoma of the head of the pancreas, whereas with ampullary growths mild epigastric discomfort may herald the onset of jaundice but does not persist. Most authors agree with this view. It is described as variable in position, type, and degree. In this series 22 patients had pain in the form of vague epigastric discomfort, severe right-sided or central epigastric pain, biliary colic, or backache. Three of these cases had chronic cholecystitis and gall-stones. We were not able to find any distinctive feature in the type of pain described by our patients.

Itch.—This symptom has received scant attention. It is usually described as a sequel of obstructive jaundice. In this series the incidence of itch was very variable. In most cases it coincided with the onset of jaundice and was severe and persistent as the jaundice deepened. In some cases, however, it disappeared after a few days and did not return even though the jaundice progressed. In each of the 8 cases of ampullary tumour generalized itching preceded the appearance of jaundice by an interval of from two to six weeks, and was the first symptom noted by the patient. Illingworth (1951) describes a case in which itching occurred for three months before jaundice appeared, the patient having a small malignant ulcer on the duodenal aspect of the ampulla. He suggests that the obstruction of the bile-duct was sufficient to raise the bile-salt in the blood to the ' itching level ' without raising the bile-pigments enough to produce clinical jaundice. The assumption that excess of bile-salts causes itching is not generally accepted. Itching also occurs in jaundice due to other causes, so that it does not appear to be of much diagnostic value except that, when it precedes the onset of obstructive jaundice by several weeks, it suggests that the site of obstruction is the ampulla.

However desirable it may be to make the diagnosis before jaundice has occurred, this aim is seldom achieved. Symptoms arising before icterus will depend largely on the site of origin of the tumour. For example, if the pancreatic duct is obstructed early the only symptoms may be vague dyspepsia, alteration in bowel habit and character of the stools, and backache. These symptoms are vague and non-specific, and are similar to those due to carcinoma arising in the body or tail of the pancreas, and it is significant that the diagnosis of growths in those situations is seldom made at an early or operable stage.

Examination.—Probably the most important finding in the clinical examination is a palpable gall-bladder. In this series it was palpable in 29 cases, or 63 per cent (*Table III*). It is generally accepted that it can be palpated in 60–70 per cent of cases. When clearly palpable in a jaundiced patient it is almost diagnostic of neoplastic obstruction of the common duct, since in cholelithiasis the gall-bladder is nearly always too thick walled to be distended by back-pressure. Possible exceptions to this statement are cystic duct obstruction (mucocele of the gall-bladder) by a stone which also compresses the

common duct sufficiently to cause jaundice, or mucocele associated with jaundice of some independent cause.

The liver is almost invariably enlarged and sometimes tender. We were able to feel a mass, separable from the liver and gall-bladder, in 5 of our cases and, as might be expected, each proved to have a large inoperable growth in the head of the pancreas. When the pancreatic duct is obstructed, the pancreas is found at operation to be enlarged and firm, but we have not been able to palpate it clinically.

Table III.—STATE OF THE GALL-BLADDER

Palpable		29 (63 per cent)
Impalpable		17 (37 per cent)
Found distended at operation	15	
Small and full of gall-stones	2	
Previously removed		3

The stools are usually putty coloured, although pigment may appear in them at times if local conditions in the tumour allow bile to leak temporarily past the obstruction in the bile-duct. The urine contains bile-pigment once jaundice has developed, and in 3 of our cases contained urobilinogen in normal amounts, but never in excess. In the remainder urobilinogen was absent.

INVESTIGATIONS AND DIAGNOSIS

When a patient presents symptoms such as those described as pre-icteric in this series, the diagnosis of carcinoma arising in the pancreas should be borne in mind. The examination and investigation of such a case will naturally be along the usual lines, but tests which may assist in locating the tumour in the pancreas are occult blood examinations, duodenal intubation, the secretin test, and radiology. Occult blood may be present in the stools in cases of ampullary carcinoma or carcinoma of the head of the pancreas which is invading the duodenum. It is less likely to be detected when the growth is confined to the pancreas. In this series frank melæna occurred in 3 cases, in each of which the duodenum was involved. Duodenal intubation aims at the demonstration of blood, mucus, and malignant cells in the fluid removed. When combined with the injection of secretin, the composition of pancreatic secretions may be studied. Parsons (1951) concludes that the demonstration of cancer cells is likely to be successful if ulceration is present, but that the study of pancreatic ferments is not helpful. Rodney Smith (1953) shows that skilled radiology is capable of demonstrating tumours of the ampulla and tumours in the head of the pancreas provided that some invasion or distortion of the duodenum has occurred.

When jaundice is present the problem becomes one of distinguishing between pure obstruction and prolonged hepatitis. Either may be ushered in by a gastro-intestinal disturbance, and either may be painless. An enlarged tender liver may be found in both, together with dark urine and pale stools. Unless the case is seen early the phase of excessive excretion of urobilinogen in the urine in hepatitis may have passed, but if it is still present it excludes pure obstruction.

We have found tests of liver function of great value, and out of the large number available have used only a few as routine procedures, namely, the total and differential plasma-proteins, the thymol turbidity test, the gold and thymol flocculations, and the alkaline phosphatase. In our cases of pure obstruction without secondary liver damage the flocculations have been invariably negative and the thymol turbidity (normal 0–4 units) never above 4 units.

The alkaline phosphatase is raised early in obstructive jaundice and late in severe hepatitis. In early cases, therefore, a high figure indicates obstruction and a dividing line of 30 units (normal 4–13 units) has been suggested (Sherlock, 1946), but this is not clear cut. In 30 of our cases the alkaline phosphatase was over 30 (commonly 50, 60, or 70, and in 2 cases over 100), and in these, with negative flocculations, the diagnosis was regarded as virtually certain. In 17 cases, however, it was below 30, and had not risen when repeated after a short interval, but in these cases the flocculations remained negative and the serum-bilirubin increased, and this has not been observed to occur in persistent hepatitis. In a few cases the alkaline phosphatase level was found to decrease slowly while the serum bilirubin was rising.

Ascending cholangitis may give rise to difficulty. It presents the picture of obstructive jaundice, but is usually associated with rigors, or at least with fever higher than the small rise in temperature found in some cases of carcinoma of the pancreas.

We do not consider that the differential diagnosis is difficult if the patient is seen soon after the onset of jaundice. During the period in which this series of cases was seen, one case of hepatitis was incorrectly diagnosed as obstructive jaundice and laparotomy performed. At the time of operation the liver function tests were doubtful, and in the light of our later experience we would delay operation in such a case. When the tests were repeated ten days later they showed clear evidence of hepatitis.

Every case in this series, except two, was jaundiced when first seen by one of us, and the jaundice could be labelled ' obstructive ' at the first examination with the aid of liver function tests in the majority, and in the remainder after a short interval. Once the diagnosis of obstructive jaundice has been made we believe that operation should not be delayed in order that further investigations may be carried out in an attempt to reach a more exact diagnosis.

PREPARATION FOR OPERATION

It is very important to give the patient vitamin K for two or three days before operation in order to restore the plasma-prothrombin level. It may be given by mouth combined with bile-salts, or by injection. The electrolyte balance must be restored by suitable means if it is disordered, and anæmia corrected by transfusion unless it is only slight. A high protein intake should be given if the plasma-proteins are low. Breathing exercises and postural drainage of the bronchial tree are very important, especially as many of these patients are elderly.

CHOICE OF OPERATION

D'Offay (1946) has given an excellent review of the history of the development of the operation of

pancreatico-duodenectomy. An even fuller review is given by Child (1949).

In the gradual evolution of the present-day operation a great variety of methods was tried. This was largely owing to the need to find a final arrangement of the parts concerned which would function, was free from complications such as entry of stomach contents into the gall-bladder, and was so planned that part could be done as a first stage when the patient was too ill to have it all done at once, and the rest when his condition had improved sufficiently as a result of what had been done in the first operation. The first stage had to consist of anastomosis of the gall-bladder to the stomach or jejunum. This was apt to leave the surgeon with some awkward 'ends', such as the common bile-duct, 'left over' at the end of the second stage, and sometimes to result in troublesome adhesions.

Opinion as to whether a one-stage or a two-stage operation should be performed is gradually reaching uniformity and is perhaps best summarized by Cattell (1945) when he says:—

a. In most instances a one-stage procedure is preferable.

b. The criteria used as indications for a two-stage operation are: severe debility, a serum-bilirubin of 5 mg. or more, and severe impairment of liver function.

We have taken the view that if the patient's condition is not good enough to stand a single-stage operation, then the simpler the procedure at the first stage the better. We have, therefore, performed simple external cholecystostomy as the first-stage plan, and believe that its advantages over internal bile drainage are considerable. It is simple, it leaves the field of subsequent resection undisturbed (the tube is placed as far out in the flank as possible), it leaves the surgeon free to employ any form of reconstruction of the alimentary tract that he desires at the second stage, it can be easily converted to cholecyst-duodenostomy or cholecyst-jejunostomy if for any reason it is found at the second stage that resection cannot be completed, and it is free from the disadvantage of internal cholecystostomy mentioned by d'Offay in which the patient feels so much better as a result of the relief of jaundice that he refuses to undergo the second stage.

The advantages of a single operation are of course obvious; the avoidance of delay, a clean field, a single anæsthetic, and the psychological effect on the patient. These advantages are reversed in a two-stage plan, although the general condition at the time of the second stage should on average be better than in those considered fit for a single operation, since the jaundice will have been relieved.

A theoretical disadvantage of external cholecystostomy is that a disaster, such as coronary thrombosis, might occur to prevent the second stage and the patient would thus be condemned to a permanent 'tube and bottle'. We do not think that this risk should weigh very heavily against an operation which has so many advantages.

In 2 of our cases choledochostomy was performed because in one the gall-bladder had been previously removed, and in the other it was so grossly diseased that it could not be used. In several cases the gall-bladder contained multiple calculi but was only moderately thickened, and in these the calculi were removed and the cholecystostomy performed in the usual way. We believe that it is very important to preserve the gall-bladder in this disease whenever possible. Two patients were gravely ill with cholæmia when first seen. Both had large palpable gall-bladders and simple cholecystostomy was performed through a small incision without any attempt being made to explore the abdomen or establish an exact diagnosis. Both recovered and underwent successful resection later.

The loss of bile from the alimentary tract is compensated to some extent by giving bile-salts by mouth, and it is of the greatest importance that the electrolyte balance should be maintained. The progress of patients treated in this way has been satisfactory, though we have found that the serum-bilirubin may rise for a time after cholecystostomy, and no appreciable reduction in clinical jaundice is usually seen for two weeks. *Table IV* shows the behaviour of the serum-bilirubin in our two-stage cases. The table suggests that the deaths (with the exception of *Case* 12, in whom the cause of death was a pancreatic fistula) were related to the presence of jaundice and a short interval between the stages, and it seems reasonable to draw the conclusion that the interval between stages should be not less than three weeks.

Table IV.—Two-stage Pancreatectomy. Serum-bilirubin Readings in relation to Interval between Stages

Case	Initial Serum-bilirubin and Subsequent Readings	Interval between Stages	Clinical Jaundice	Result
		days		
1	20·0	22	Clear	Recovered
2	10·3	22	Clear	Recovered
6	5·7	21	Clear	Recovered
7	Not estimated	16	**Trace**	**Died**
9	13·2 . . . 2·9	37	Clear	Recovered
10	4·0	28	Clear	Recovered
11	9·2 . . . 2·6	30	Clear	**Died**
12	2·0 . . . 4·8	31	+	**Died**
14	4·2 . . . 16·0 . . . 14·7 . . . **12·3**	13	+	**Died**
15	17·3 . . . 10·8 . . . 3·0	28	Clear	Recovered
16	13·2 . . . 9·0	14	**Trace**	**Died**
30	15·8 . . . 3·0	25	Clear	Recovered
33	8·3 . . . 15·3 . . . 4·5	41	Clear	Recovered

DIAGNOSIS AT OPERATION

The first problem confronting the surgeon on opening the abdomen is that of making a firm diagnosis which will justify the major steps he is about to take. All writers stress this difficulty.

Tumours of the ampulla can be directly inspected by opening the duodenum, and immediate frozen-section biopsy may be done if doubt remains. Benign tumours of the ampulla have been recorded, but are extremely rare (Child, 1949).

Tumours of the lower end of the bile-duct may closely resemble a stone when palpated through the surrounding tissues. When the common duct is opened and a probe is passed down to the obstruction and pressed against it, biliary mud and portions of a stone may escape and proclaim the diagnosis, but this procedure may detach malignant cells from a carcinoma of the duct and there is at least a theoretical danger of their implantation in the operation field. If it is impossible to make a decision by palpation alone, and the surgeon feels that he must open the bile-duct, the opening should be sited carefully so as not to interfere with the use of the duct for anastomosis later, and on no account should the fluid in the duct be allowed to escape on to the surrounding tissues.

When the biliary tract and the remainder of the pancreas are healthy, it is unlikely that a small area of pancreatitis will be found capable of completely obstructing the bile-duct, and in such a case a small constricting neoplasm is almost the only possible diagnosis. When cholelithiasis is present, or the history includes previous attacks of jaundice, the possibilities are wider, as the following two cases illustrate.

Female, aged 62, seen in attack of typical cholecystitis. No jaundice, but jaundice had been observed in previous attacks. X rays showed multiple gall-stones. At operation, gall-stones in contracted gall-bladder, common duct not dilated, small hard lump felt just deep to ampulla. Duodenum opened, ampulla normal, lump just deep to it. Gall-bladder opened, stones removed, probe passed through cystic duct down common duct, but would not pass lump to enter duodenum. External cholecystostomy, enlarged lymph-gland removed for biopsy. Biopsy showed reactive hyperplasia only. Second operation two weeks later with full preparation for pancreatectomy. Lump as before. Considered insufficient evidence to justify radical operation. Cholecyst-duodenostomy. This patient has remained fit and well for three years (except for developing a primary carcinoma of the breast treated by radical mastectomy).

Female, aged 63, flatulent dyspepsia, attacks of epigastric pain and vomiting, pains R. shoulder, all for 6–9 months. X rays showed multiple gall-stones. Jaundice appeared two days before operation. At operation, gall-stones in contracted gall-bladder, common duct dilated, small area of vague thickening in head of pancreas. Gall-bladder opened, stones removed, probe passed through cystic duct down common duct but would not enter duodenum. Duodenum opened, ampulla normal. Cholecyst-duodenostomy. Patient remained well for three months, then developed abdominal pains, constipation, anorexia, and loss of weight. Operation, hard lump in head of pancreas obviously malignant, no secondaries, but radical operation abandoned because mesenteric vessels frozen. Death two months later. Biopsy of local lymph-gland removed at second operation —' replaced by adenocarcinoma '.

The essential difference between these two cases is that in the second case the common bile-duct was dilated. Cattell stresses this point and states that in his cases of cancer of the head of the pancreas the common duct was always dilated, and he concludes that the radical operation (when malignancy cannot be proved at the operating table) should not be done unless it is.

When larger tumours are found in the head of the pancreas there may be great difficulty in differentiating neoplasm from chronic pancreatitis. Either may be localized or diffuse, and both may be very hard. A scirrhous type of growth may show characteristic retraction in one or other aspect or may be commencing to invade the duodenum. When the pancreatic duct is obstructed the body and tail of the pancreas are much firmer than normal and may be swollen, but retain their natural shape and have not been in our experience as hard or as irregular as the region of the growth. However, chronic pancreatitis of any degree from mild thickening to cavitation and frank abscess formation, or even calcification, may be found, and a small carcinoma in such a gland may well be impossible to detect.

Direct biopsy of the suspected part may fail to obtain a portion of the growth and there is at least a theoretical objection to incising directly into the growth even if the surgeon is prepared to proceed to pancreatectomy there and then. Biopsy through the duodenum, as described by Rodney Smith (1953), certainly seems safer than biopsy through the anterior surface of the gland.

Carlson (1950) discusses the difficulty of establishing the diagnosis of tumours in the head of the pancreas at operation and describes 3 cases in which radical operation for non-malignant conditions might well have been carried out on clinical and operation findings only. He concludes that radical operation should not be undertaken unless histological proof of carcinoma is obtained, and stresses the operative mortality of the radical procedure (20–60 per cent) compared to a palliative operation (Cattell, 0 out of 22 recent cases) and the small extra length of average survival of radical operations compared to palliative ones (quoting Cattell, 11 months as against 9·2 months).

We consider that this attitude is unnecessarily gloomy. In many cases a confident diagnosis can be made by palpation alone. We have not used frozen-section biopsy in this series, yet every case submitted to radical operation has been proved malignant, and no case has been diagnosed malignant at operation incorrectly. Biopsy should have been performed in one patient in whom an early carcinoma was missed. We firmly believe in the principle of obtaining histological proof of malignancy before performing any form of radical operation whenever possible, but in this instance the obtaining of positive biopsy material may not always be successful, and furthermore may introduce dangers of its own. Biopsy should only be done in doubtful cases, and if it fails to show carcinoma the right course is undoubtedly a palliative operation.

OPERABILITY

The next difficulty facing the surgeon is the operability of the growth. The interesting anatomy of the relationship of the pancreas to the portal vein

provides perhaps the most anxious moment in the radical operation. Tumours which arise near the neck of the pancreas, or in the uncinate process, may involve the portal vein or superior mesenteric vessels before they involve the bile-duct. For this reason a case with a short history and relatively small tumour may be inoperable solely because the superior mesenteric vessels are 'frozen'.

When the tumour lies in this region a careful investigation of the superior mesenteric vein should be done before any major step is taken. A constant large vein which collects tributaries from the anterior aspect of the head of the pancreas joins the superior mesenteric vein from the right side just at the lower border of the neck of the pancreas. Dissection here is very delicate and it is necessary to be able to ligate this vein flush with the superior mesenteric if resection is to be possible.

Attempts to separate the gland from the main vein are usually made first and sooner or later a tear may occur with severe bleeding, the controlling of which results in harm to the patient's condition and to a surgeon who has lost much of his freshness and still has much to do (d'Offay, 1946). (*Cases* 7 and 31, *Table VI.*)

Any surgeon undertaking this operation must decide whether he is prepared to resect part of the mesenteric (or portal) vein when its involvement is the only 'inoperable' feature. If so, an early decision to do so will save time and perhaps the results of a brisk hæmorrhage. When a short segment of vein is removed repair by end-to-end anastomosis is possible (Martin, 1951), but portocaval anastomosis may be the only way of dealing with a large gap.

Skirting past the portal vein, as Child (1949) points out, is one of the weaknesses of the radical operation, since the vein may lie within a millimetre or two of growth. Moreover, handling of the growth during resection may liberate malignant emboli into the rich venous plexus which drains the pancreas.

Another weakness of the radical operation is the failure to remove the whole pancreas. However small and well defined the growth in the head may appear to be, it is impossible to determine how much of the gland to remove and how much may safely be left. Sections through the divided edge of the pancreas were unfortunately not made in all the specimens in this series, but out of 14 so examined growth was demonstrated in 4, and in 3 of these it was thought that a sufficient margin of healthy pancreas was being removed. In the fourth it was suspected that the resection might be inadequate, but in this case, for other reasons, radical resection had already been abandoned. Thus, in 3 cases out of 14, so-called radical operation was doomed to failure from the very beginning.

Parsons (1951) describes the finding of cancer cells in the fluid from an obstructed pancreatic duct, and also satellite foci of cancer in the distal part of the pancreatic duct. The prognosis of patients who survive the radical operation is discussed later, but most authors with extensive experience are disappointed at the high incidence of early recurrence, and Cattell and Pyrtek (1949), after reviewing 30 cases which survived the operation for carcinoma of the head of the pancreas, conclude: "... in the light of present experience we must modify the operative procedure or abandon radical surgery ... we wish to suggest total pancreatectomy. ..."

The metabolic effects of total pancreatectomy are considerable, and discussion of them is beyond the scope of this paper, but reports are not entirely unfavourable (Whitfield, Gourevitch, and Garfield Thomas, 1952). A more extensive removal of the pancreas, with the more thorough removal of lymph-glands which this entails, certainly seems to demand

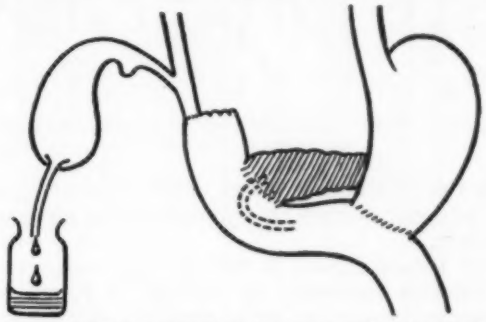


FIG. 683.—Radical pancreatico-duodenectomy as described by Whipple (1946), but with addition of external cholecystostomy.

trial, and every precaution must be taken to avoid spilling fluid, which may contain cancer cells, from the pancreatic or bile-ducts, or even the jejunum.

THE OPERATION

Space does not permit a description of the steps by which the pancreas and related parts are resected. In this series the tissue removed has consisted of the gastric antrum, the pylorus, the entire duodenum with the first 4–6 inches of jejunum, the enclosed pancreas up to a variable point of division in the region of the neck or body of the gland, together with as many lymph-glands as possible related to these structures and including glands from the portal fissure.

Reconstruction of the Alimentary Tract.—The plan for reconstruction which we have used, except in two cases in which urgent modification was necessary, is that ascribed to Whipple, 1946 (*Fig.* 683), but with the addition of external cholecystostomy either already present as a first-stage operation, or as the first step in a one-stage operation.

The Common Bile-duct.—Nearly all authors agree that it is unsafe to ligate the common duct and perform cholecyst-jejunostomy. The ligature on the duct is very liable to slough away. Furthermore, as mentioned by Rodney Smith, the ligature may inadvertently include a low joining cystic duct and the cholecyst-jejunostomy thus fail. Anatomical variations in the level of junction of the ducts are found (*Fig.* 684).

Fig. 684, (**a**) shows the commonest arrangement and presents no difficulties.

The arrangement shown in *Fig.* 684, (**b**) occurred in 6 cases, there being two separate ducts within a common sheath at the level of division. This makes the anastomosis difficult, but the difficulty is overcome by carefully excising the common septum for about half an inch (*Fig.* 685). This gives a single wide duct and the gall-bladder is thus preserved.

Fig. 684, (**c**) shows an arrangement found in 3 cases, the ducts being so separated that sacrifice of

the gall-bladder was unavoidable, since the cystic duct was too small for anastomosis.

The cholecystostomy drains about 12 oz. of bile in the first 24 hours, and thereafter diminishing amounts until the tube is removed on the 10–12th day, whereupon it promptly dries up. We feel that

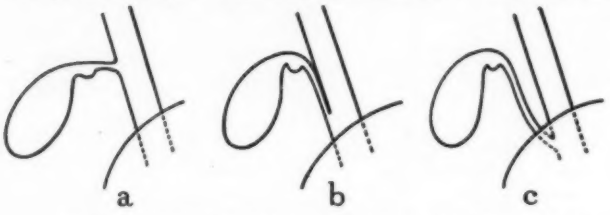


FIG. 684.—Three varieties of anatomy of junction of cystic and common bile-ducts.

this is a most valuable safety valve to avoid pressure at the bile-duct suture line should œdema temporarily occlude it.

The jejunal lumen is adjusted to match that of the common bile-duct by closing as much of its mesenteric border as is necessary. We have not used a tube or other support for this anastomosis.

The Stump of the Pancreas.—The disposal of this difficult 'end' has been and still is the subject of much difference of opinion. The possibilities

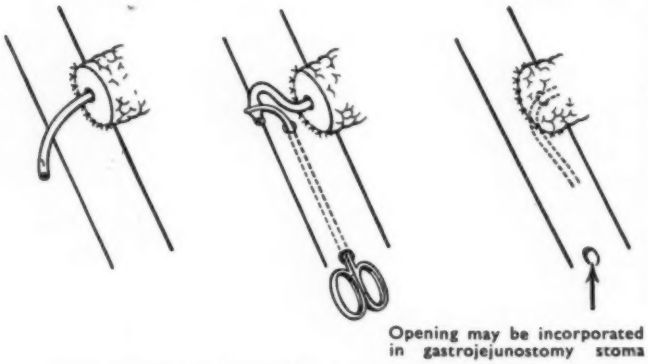


FIG. 686.—Method of implantation of pancreatic duct in jejunum by means of a rubber tube. The pancreatic capsule is united to the jejunum by interrupted sutures of fine silk or thread.

are: (1) Pancreatico-gastrostomy. (2) Pancreatico-jejunostomy. (3) Ligature duct and oversew.

A study of recorded cases shows that anastomosis is being performed more and more frequently, and the real question is as to the best method by which this may be done.

The animal experiments and the successes in human subjects obtained by Wells (Wells and Annis, 1949, Wells, Shepherd, and Gibbon, 1952) seem to show clearly that open suture of the pancreatic stump to the stomach is safer than open suture to the jejunum.

We have preferred to use the method of catheterization of the pancreatic duct with a rubber tube and implantation of this tube in the jejunum through a stab incision, as described by Whipple (*Fig.* 686).

In 2 cases in this series the stump of the pancreas was oversewn to save time when other difficulties had occurred. Both died, and post-mortem examination in one ten days later showed the oversewn stump dry and firm. In 1 case the pancreatic duct was extremely small and no suitable tube was available. Open anastomosis of the pancreatic stump to the jejunum was performed and, as in the cases described by Wells, fistula and death resulted. In the remaining 26 cases the rubber tube method was used, and in only one did a fistula occur, and it also

FIG. 685.—Method of converting unjoined cystic and common bile-ducts in common sheath into a single duct for purpose of anastomosis, thus preserving the gall-bladder.

led to death. A possible explanation of this fistula is that this was the only case in which we have found an accessory pancreatic duct in the divided stump. The accessory duct was very small and was oversewn while the main duct was catheterized in the usual way. Leakage from the accessory duct may have led to the breakdown of the anastomosis and the fistula which resulted.

The Jejunal Loop.—We have removed the upper 4–6 in. of jejunum in order to obtain long jejunal arteries which will permit the open cut end of the jejunum to reach the porta hepatis without suspicion of tension. In our early cases the jejunum was brought up through the opening in the transverse mesocolon created by the removal of the duodenojejunal flexure and so lay beneath the superior mesenteric vessels. No untoward effect was noted in the first 6 cases, but the seventh case developed intestinal obstruction which necessitated re-opening the abdomen on the fifth day. The site of obstruction was found to be where the jejunal loop passed beneath the mesenteric vessels and was relieved by gastro-enterostomy (*Case* 10). Since that time the jejunum has been brought up in front of the mesenteric vessels through an incision in the transverse mesocolon to the right of the mid-colic vessels.

Table V.—RADICAL PANCREATICO-DUODENECTOMY

	SITE	OPERATIONS	RECOVERED	DIED
One-stage	Ampulla	2	2	0
	Head of pancreas	13	7	6
	Duodenum	1	1	0
		Total 16	10	6
Two-stage	Ampulla	6	5	1
	Head of pancreas	7	3	4
		Total 13	8	5
Total		29	18	11

RESULTS

OPERABLE CASES—RADICAL OPERATION

Twenty-nine cases were considered suitable for radical operation (*Table V*); 18 cases survived the operation and 11 died, so that the operative mortality

Table VI.—SUMMARY OF 29 RADICAL PANCREATICO-DUODENECTOMIES

Case No.	Sex and Age	Intercurrent Disease	Operation	Site	Type	Degree of Malignancy	Growth in Cut End of Pancreas	Glands	Operation Death	Death After	Survival	Cause of Death	Remarks
1	M. 38	—	Stage 2	Head	Adenocarcinoma	Average	Not examined	None	—	Months —	Months 56	—	—
2	F. 66	—	2	Head	Adenocarcinoma	Low	Not examined	None	—	15	—	Clinical secondaries	—
3	F. 66	—	1	C.B.D.	Adenocarcinoma	Average	0	1–4	21st day	—	—	Jejunal fistula	—
5	F. 73	Gall-stones	1	Head	Adenocarcinoma	Average	Not examined	0–1	14th day	—	—	Pancreatic fistula	—
6	F. 68	—	2	Ampulla	Anaplastic	High	Not examined	1–1	—	6	—	Clinical secondaries	—
7	F. 58	—	2	Head	Adenocarcinoma	Average	Not examined	0–5	48 hr.	—	—	Circulatory failure	Resection portal vein
9	F. 84	Gall-stones	2	Head	Adenocarcinoma	Average	+	0–2	—	12	—	Clinical secondaries	—
10	M. 58	—	2	Ampulla	Papillary adeno-carcinoma	Average	Not examined	None	—	—	33	—	—
11	F. 61	—	2	Head	Adenocarcinoma	High	Not examined	None	48 hr.	—	—	Circulatory failure	—
12	F. 63	—	2	Ampulla	Adenocarcinoma	High	Not examined	2–6	20th day	—	—	Pancreatic fistula	—
14	F. 78	Chronic nephritis	2	Head	Adenocarcinoma	Average	Not examined	None	8th day	—	—	Anuria	—
15	F. 79	—	2	Ampulla	Papillary adeno-carcinoma	Average	Not examined	1–3	—	—	30	—	—
16	M. 74	Diabetes	2	Head	Adenocarcinoma	Low	0	None	26th day	—	—	Liver failure	—
17	M. 71	Parkinsonism	1	Head	Adenocarcinoma	Average	Not examined	2–2	20th day	—	—	Hæmatemesis (acute G.U.)	—
22	M. 69	—	1	C.B.D.	Adenocarcinoma	Average	Not examined	2–5	—	—	25	—	—
26	F. 73	—	1	C.B.D.	Adenocarcinoma	Average	0	2–4	—	—	20	—	—
27	M. 76	Bronchitis, cholangitis	1	Head	Adenocarcinoma	Average	+	0–1	13th day	—	—	Cholæmia	—
28	F. 60	—	1	Ampulla	Papillary adeno-carcinoma	Low	0	0–3	—	—	19	—	—
30	M. 60	—	2	Ampulla	Papillary adeno-carcinoma	Low	0	0–6	—	—	15	—	—
31	F. 67	—	1	Head	Adenocarcinoma	Average	+	0–3	10th day	—	—	Liver necrosis	Resection portal vein; porto-caval anastomosis

Note.—In the column headed 'Glands', the figures 1-4 mean 1 gland metastasis out of 4 examined. 'None' means that no glands were found in the specimen removed.

[*Continued*

Table VI.—Summary of 29 Radical Pancreatico-duodenectomies, *continued*

Case No.	Sex and Age	Intercurrent Disease	Operation	Site	Type	Degree of Malignancy	Growth in Cut End of Pancreas	Glands	Operation Death	Death After	Survival	Cause of Death	Remarks
32	F. 47	—	Stage 1	Duodenum	Adenocarcinoma	Average	Not examined	0–4	—	Months 8	Months —	Proved secondaries	—
33	F. 57	—	2	Ampulla	Mucoid adeno-carcinoma	Low	0	1–2	—	—	12	—	—
34	M. 63	Enlarged prostate	1	Head	Adenocarcinoma	High	0	0–3	—	—	13	—	—
36	F. 60	Pancreatic calculi	1	Head	Adenocarcinoma	Average	+	2–5	3rd day	—	—	Circulatory failure	—
38	F. 66	—	1	Head	Specimen inadvertently destroyed				—	5	—	Proved secondaries	Biopsy: single liver nodule = adeno-carcinoma
42	F. 74	—	1	Ampulla	Adenocarcinoma	Average	0	0–4	—	—	7	—	—
46	M. 80	—	1	Head	Adenocarcinoma	Average	0	0–6	—	—	5	—	—
47	M. 61	Cerebral throm-bosis	1	C.B.D.	Adenocarcinoma	Average	0	0–8	—	—	4	—	—
48	F. 73	—	1	Head	Adenocarcinoma	Average	0	0–4	—	—	3	—	—

Note.—In the column headed 'Glands', the figures 1–4 mean 1 gland metastasis out of 4 examined. 'None' means that no glands were found in the specimen removed.

was 38 per cent. More detailed results are given in *Table VI*.

Operation Deaths.—These number 11, 8 occurring in the first 14 operations, only 3 in the next 15. Because this figure is high, a detailed analysis of the cause of death is given in *Table VII*.

The mortality-rate for any major operation is closely related to the standards of operability of the individual surgeon as well as to his technical skill, particulary so in this type of case. If the radical operation is reserved for the younger patient, fit in other respects, not deeply jaundiced and with a small tumour, a high recovery rate is to be expected, but the operability rate will be low. The alternative to radical operation is a palliative operation designed merely to relieve jaundice, leaving the carcinoma to advance unchecked into the surrounding tissues, and it is doubtful whether the term 'palliative' should be used for such operations at all. In this series the standards of operability have been both optimistic and enthusiastic, as consideration of *Table VII* will no doubt show, and our reasons for attempting radical operation whenever possible were firstly, the miserably short benefit which palliative procedures give (*Table VIII*), which is discussed later, and secondly the fact that the radical operation, compared to other major abdominal operations, is as yet relatively untried. It is interesting to note that, in the matter of age limit, the three oldest patients who underwent radical operation, aged 79, 80, and 84 respectively, all made uneventful recoveries.

Recovery from Operation, since died.—Five cases which survived the operation died 5, 6, 8, 12, and 15 months later, respectively, and in each death was due to clinical carcinomatosis. In two of these the outcome was certain, since in one histological examination of the specimen removed showed growth present in the cut edge of the pancreas, and in the other a single tiny nodule removed from the liver for biopsy showed carcinoma. One case (*Case* 32, *Table VI*) is of especial interest. The primary in this case was a large ulcer of the medial wall of the second part of the duodenum.

Case 32.—Female, aged 47, one-stage radical operation with uneventful recovery. Report on specimen: "A shallow ulcer in the second part of the duodenum measuring 3 × 5 cm.; its base is formed by the pancreas, and the ampulla has been completely destroyed. Sections from the edge of the ulcer show infiltration by a poorly differentiated adenocarcinoma; the pancreas forming its base is free from growth, as are the termination of the bile and pancreatic ducts. No invaded glands can be found. I do not think it is possible to be certain whether the growth arose from the ampulla or from the duodenal mucosa; on the whole the appearances and the histology favour the latter." (Dr. A. Clay.)

After good progress for three months, recurrence of jaundice and spontaneous re-opening of cholecystostomy. Operation: carcinomatous involvement of jejunal loop used in the reconstruction, especially in relation to the sites of the three anastomoses. No evidence of carcinoma found elsewhere. Cholecyst-jejunostomy the only possible palliation. Temporary relief of jaundice. Death 8 months after original operation.

The early and unexpected recurrence of growth in this case led us to suspect, in view of the sites in which it was found, that it might have been the result of direct implantation of malignant cells from

the lumen of the jejunum on to the raw suture lines of the anastomoses, such as has been shown to occur by Goligher, Dukes, and Bussey (1951) in cases of conservative resection of the rectum.

Recovery from Operation, alive.—Thirteen cases survived the operation and all were well and free from signs of recurrence when last seen. One of these, who had a carcinoma of the head of the pancreas, has survived 56 months.

INOPERABLE CASES—PALLIATIVE OPERATION

Eighteen cases out of 49 were considered to be inoperable, and in a further 2 cases cholecystostomy with a view to possible radical operation was followed by death. This gives an operability rate of approximately 60 per cent. Cattell and Pyrtek (1949), reviewing 165 patients in the Lahey Clinic between 1942 and 1948, found that 56 had undergone the radical operation, i.e., almost exactly one-third.

Table VII.—Operation Deaths

Case	Age	Cause of Death	Comment
3	66	Jejunal fistula. 21st day	Complete fistula developed suddenly on 8th day, on removing drainage-tube. Opening found in jejunum not related to any anastomosis. Repair and jejunostomy. Fistula recurred, death 21st day. Perforation possibly due to pressure of drainage-tube, possibly acute jejunal ulcer. At post mortem anastomoses intact
5	73	Pancreatic fistula. 14th day	Open anastomosis of pancreatic stump to jejunum. Inadequate method of dealing with pancreatic stump
7	58	Shock, circulatory failure. 48 hours	Tumour adherent to portal vein. Tearing of vein, resection of one inch portal vein and end-to-end anastomosis. Blood-loss, shock, and delay. Vein anastomosis did not leak or thrombose
11	61	Shock, circulatory failure. 48 hours	Poor risk patient. Obese. Atheroma of coronary arteries
12	63	Pancreatic fistula. 20th day	Operation apparently satisfactory. No explanation for fistula except possibly initiated by leakage from oversewn accessory pancreatic duct
14	78	Anuria. 8th day	Operation uneventful. Anuria from 1st day. Post mortem showed advanced renal degeneration, operation area healing
16	74	Asthenia, diabetes, liver failure	Poor risk patient. Chronic suppurative pancreatitis
17	71	Hæmatemesis from multiple acute gastric erosions. 20th day	Parkinson's disease. Radical operation not intended, but head of pancreas so mobile that operation technically very easy and therefore preferred to palliative procedure. Smooth recovery until sudden collapse and death at once
27	76	Cholæmia, cholangitis. 13th day	Poor risk. Suppurative cholangitis found at operation
31	67	Asthenia, liver failure. 10th day	Tumour invading portal vein. 2 in. portal vein resected. Splenic vein tied. Superior mesenteric vein anastomosed to inferior vena cava, stump of portal vein tied. At post mortem vein anastomosis patent, other anastomoses intact, no peritonitis, no mesenteric thrombosis. Sections of liver showed extensive centrilobular necrosis, kidneys showed well-marked tubular necrosis
36	60	Shock, circulatory failure. 48 hours	Previous cholecystectomy, much fibrosis. Malignant extension in posterior abdominal wall unrecognized until late in operation. Case strictly inoperable.

Palliative operations aim at relief of jaundice (and the usually accompanying itch), or occasionally duodenal obstruction. The procedures include cholecyst-duodenostomy, and cholecyst-jejunostomy, with or without gastro-jejunostomy.

Nearly all authors give the average period of survival after palliative operations as nine months. Of our 18 cases, five failed to survive the operation, 8 survived an average of five months, and 3 recent cases are still alive. The longest survival was *Case* 35 in which, owing to misdiagnosis, cholecyst-duodenostomy was performed when the tumour was at an essentially operable stage. Yet death occurred after 11 months. With this case excepted, the longest survival after palliative operation has been a mere 25 weeks (*Table VIII*).

It is of course clear that if borderline cases are submitted to radical operation, those deemed inoperable will be advanced cases in whom long survival after palliative operations can hardly be expected. Yet if the mortality-rate for radical operation appears formidable and unjustifiable, and if the successful radical operation gives an average survival of 11 months (Cattell and Pyrtek, 1949), which is not significantly longer than that of simple palliative operation (nine months), and if palliative operation does not prolong life beyond the average period of 6–8 months which is the natural course of the untreated case of obstruction of the bile-duct by cancer of the ampulla or head of the pancreas (Rodney Smith, 1953, quoting Oughterbridge, 1913, Lieber, Stewart, and Lund, 1939), then the conclusion must be reached that surgery has no more to offer for this disease than the relief of jaundice.

The results in the 18 cases in which palliative operations were done in this series are summarized in *Table VIII*. They were nearly all relatively advanced cases, and although most showed clinical improvement for a time, it was short lived, and the early optimism which the operation brought was

quickly replaced by bitter disillusionment. In fact, so depressing are some of these cases that it is questionable in our opinion whether death from unrelieved cholæmia is not more merciful than the efforts of the surgeon to prolong the time in which pain and other symptoms may gather force.

CARCINOMA OF THE AMPULLA

So far we have drawn little distinction between carcinoma of the ampulla and carcinoma arising in the head of the pancreas, apart from certain clinical considerations. There were 8 cases of ampullary carcinoma in this series and each was treated by radical operation. However, simple transduodenal removal of the tumour has been described and successfully performed by many surgeons, from Halsted (1899) onwards. In 1941 Hunt summarized 93 published cases. It is a temptingly simple and safe procedure compared to the radical operation in a patient doubtfully fit to undergo the latter, and it is probably correctly chosen in certain cases. Long survival following this operation has been reported (Brunschwig, 1942).

Table IX gives the histological reports on the specimens removed by radical operation in the 8 cases in this series and indicates the prognosis which could have been expected after local resection of the tumours.

The mortality-rate for the radical operation is much lower for carcinoma of the ampulla than for carcinoma of the head of the pancreas. The probable reasons for this are that the former produce jaundice earlier, become 'fixed' later, and are remote from planes of dissection and vital blood-vessels. The largest published series is that of Cattell and Pyrtek (1949), which shows an operative mortality of 5 per cent in 20 resections for the former compared with 16·5 per cent in 30 resections for the latter. In this series the contrast is even more striking, being 1 death out of 8 cases for the former, and 10 out of 21 cases for the latter.

The difference in long-term survival is also striking. In Cattell's series 6 out of 12 ampullary cases followed up were alive after three years. Only 7 out of 25 cases of carcinoma of the head of the pancreas were still alive and only 1 of these had passed the three-year mark; the average survival of those already dead was eleven months. Our series is too small, and many of the cases too recent for conclusions to be drawn.

Table VIII.—Summary of 20 Inoperable Cases

Case	Sex	Age	Reason for Inoperability	Operation	Death	Died (weeks)	Alive (weeks)
4	F.	74	Liver secondaries	Cholecyst-jejunostomy	4th day—cholæmia		
8	M.	60	Liver secondaries	Cholecyst-duodenostomy Gastro-jejunostomy		14	
13	M.	72		Cholecystostomy, for possible later radical	21st day—liver failure		
18	F.	55	Fixity	Cholecyst-jejunostomy	7th day—hæmatemesis acute gastritis		
19	F.	52	Fixity	Cholecyst-jejunostomy Gastro-jejunostomy		10	
20	F.	37	Liver secondaries	Cholecyst-duodenostomy		20	
21	M.	62	Fixity	Cholecyst-jejunostomy		20	
23	F.	63	Fixity	Cholecyst-jejunostomy Gastro-jejunostomy	20th day—liver failure		
24	M.	72	Fixity	Cholecyst-jejunostomy Gastro-jejunostomy	3rd day—coronary thrombosis		
25	M.	65	Operable, but cardiac aneurysm	Cholecyst-duodenostomy		24	
29	M.	68	Liver secondaries	Nil possible		7	
35	F.	63	Misdiagnosis, later, fixity	Cholecyst-duodenostomy		48	
37	F.	81		Cholecystostomy, for possible later radical	7th day—cholæmia		
39	F.	72	Fixity	Cholecyst-duodenostomy		25	
40	M.	66	Fixity	Cholecyst-duodenostomy		22	
41	M.	70	Fixity	Cholecyst-duodenostomy	42nd day—coronary thrombosis		
43	M.	61	Involvement of mesenteric vein	Cholecyst-duodenostomy			22
44	M.	69	Liver secondaries	Cholecyst-jejunostomy Gastro-jejunostomy		12	
45	F.	42	Liver secondaries	Cholecyst-duodenostomy			15
49	M.	53	Involvement of mesenteric vessels	Cholecyst-duodenostomy			4

CONCLUSIONS

In the operation of pancreatico-duodenectomy a very considerable alteration of physiology and function is produced. The pylorus, the sphincter of Oddi, the duodenum, and about half the pancreas are taken away. Yet the published results show that the patients who survive can live normal lives, unrestricted in diet and habit. This is more than can be said for some of the radical abdominal operations successfully performed nowadays for cancer.

The mobilization and removal of the head of the pancreas and the anastomoses which follow are not difficult feats when the organs concerned are healthy. They become intensely difficult and dangerous when the pancreas is the seat of a large cancer adherent to vital structures. Abdominal cancer all too frequently reaches an advanced stage before symptoms can be recognized and its presence suspected. But in this disease jaundice often occurs when the disease is, as it were, in its infancy. It is vitally important that this most valuable sign should be heeded as soon as it appears, so that operation may be done before the jaundice becomes so deep that a two-stage operation is necessary, with the delay it entails, and before the growth has advanced so far as to make its removal hazardous or impossible. In this series the average duration of jaundice when first referred for investigation was just over three weeks (excluding one case in which jaundice had been present intermittently for ten months).

Tests of liver function are more reliable in the early stages than later, when liver damage is caused by back-pressure. We do not claim that every case of jaundice can be correctly diagnosed within a short time of its onset, but a careful assessment of all the clinical and laboratory evidence will point the way in most cases.

A recent author has stated: "I make a plea that in cases in which painless jaundice has been present for a month the possibility of carcinoma of the pancreas should be considered and a surgical opinion sought."

While agreeing with him wholeheartedly, we would rather say: "In every case in which jaundice, whether painless or not, occurs in a patient over 30 (or even younger), the possibility of carcinoma of the pancreas should be considered, and a full investigation by all available means should be carried out with the least possible delay".

Table IX.—HISTOLOGY OF 8 CASES OF CARCINOMA OF AMPULLA (DR. R. CLAY, DEPARTMENT OF HISTOLOGY, CENTRAL LABORATORY, PORTSMOUTH)

Case 6.—Spheroidal cell carcinoma; some signet-ring cells. Infiltrating muscle coats of adjacent duodenal wall, but no invasion of underlying pancreas. Supra-pyloric gland contains a large deposit of growth.

Case 10.—Papillary columnar-celled carcinoma infiltrating the underlying pancreas. No invaded glands were found. (*By courtesy, Dr. L. W. Proger, Pathological Curator, Royal College of Surgeons, London.*)

Case 12.—Ulcerated adenocarcinomatous nodule replacing ampulla. Invasion of duodenal wall but not of underlying pancreas. One lymph-node replaced by growth.

Case 15.—Papilliferous adenocarcinoma blocking common duct at ampulla and projecting into duodenum, invading underlying pancreas. One gland showed metastasis.

Case 28.—Papillary adenocarcinoma of ampulla. Invading adjacent duodenal muscularis. No metastasis in three glands examined.

Case 30.—Columnar-celled adenocarcinoma replacing ampulla, invading wall of duodenum, but not the underlying pancreas. Three glands show no metastasis.

Case 33.—Papillary adenocarcinoma of ampulla invading duodenal wall but not the underlying pancreas. One lymph-gland shows a small metastasis.

Case 42.—Well-differentiated adenocarcinoma arising at ampulla, mainly glandular but in places papillary. Pedunculated, but invading muscle wall of ampulla, but not the underlying pancreas. No gland metastasis found.

SUMMARY

A review of 49 cases of carcinoma arising in the ampulla of Vater or head of the pancreas is presented.

Twenty-nine radical pancreatico-duodenectomies have been performed with an operative mortality of 38 per cent.

A brief description of symptoms, signs, investigations, and differential diagnosis is given.

The operative procedure which we have employed is described, and external cholecystostomy is suggested as the first stage in a two-stage radical operation.

The need for early diagnosis is stressed.

We are most grateful to Dr. H. B. C. Sandiford for the skill and understanding he has shown when giving anæsthetics for these long and often seriously ill cases. We have been greatly helped throughout by Dr. E. M. Darmady and his fellow workers, whose laboratory investigations played so big a part in diagnosis and treatment. And we wish also to thank our many colleagues who have so kindly referred cases to us.

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CYSTADENOMA OF THE PANCREAS

DESCRIPTION OF A CASE AND REVIEW OF SYMPTOMATOLOGY

BY DAVID H. TRAPNELL, READING

CYSTADENOMA of the pancreas is a very uncommon condition. As far as I can discover only 2 cases have been reported in this country (Carling and Hicks, 1925) and 1 case in Ireland (Pringle, 1925), while a further 2 cases described in the BRITISH JOURNAL OF SURGERY (Janes, 1936) were, in fact, both American cases. In 1926, Miller (quoted by Janes) could find only 24 cases in medical literature.

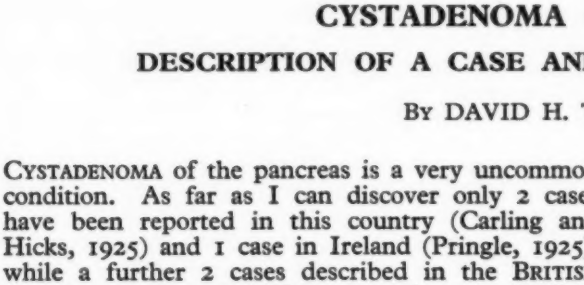

FIG. 687.—Low-power field showing cysts lined by low cuboidal epithelium and one (arrowed) partly lined by columnar epithelium. Note large area of fibrous tissue in lower right part of field. The appearances are very similar to those described by Carling and Hicks (1925) and Haukohl and Melamed (1950). H. and E. (× 50.)

In 1947, Benson and Gordon reviewed 1 case of their own and 28 cases from the literature. Since then at least another 8 cases have been described, almost entirely in the medical journals of America. In view of the apparent rarity of the condition in this country and of the difficult diagnosis and the excellent result of surgical treatment, this case is reported.

CASE REPORT

Mrs. E. R., who was 52 years old when first seen (Sept. 3, 1952), said she had noticed that she had been getting increasingly tired for about one year. For the same period she had had an aching pain in the back, in the lower thoracic and lumbar regions, which came on in the evening and lasted about two hours. She was breathless on exertion and slight swelling of both ankles was present at the end of the day, but disappeared at night. The appetite was bad, particularly for meat, and she had lost some weight recently. She had no indigestion. There was no relevant family or past history.

ON EXAMINATION.—She was pale with a slightly sallow complexion. A hard mass could be felt in the epigastrium which was just mobile and tender to pressure.

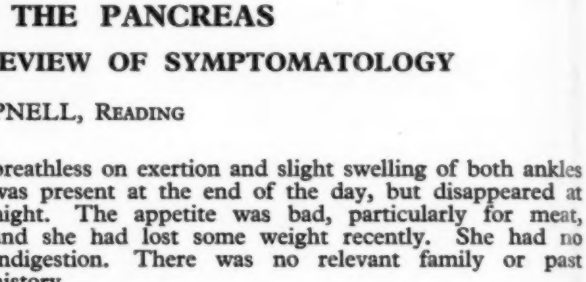

FIG. 688.—High-power field showing cyst wall and fine granular material in the cysts. H. and E. (× 400.)

The tongue was normal; there were no lymph-glands to be felt in her neck; the chest and the cardiovascular system were normal. She had a diffuse nodular mastitis in the left breast. The central nervous system was normal.

INVESTIGATIONS.—The Hb was 50 per cent, the total red cells were 4,000,000 per c.mm., colour index 0·6, while the total and differential white-cell count were normal. The red cells showed hypochromasia. There was no occult blood in the stools. A barium meal and follow-through and barium enema were all normal.

In view of the increasing lassitude, loss of appetite and weight, the sallow complexion and the abdominal mass, a provisional diagnosis of carcinoma of the stomach was made in spite of the normal radiograph. The surgeon agreed with the diagnosis and suggested laparotomy.

AT OPERATION (Mr. G. Bohn).—The abdomen was opened by an upper midline incision and a polycystic tumour the size of an orange was seen in the lesser sac. It replaced the tail and body of the pancreas and was

attached to the head by a thick pedicle which was ligated in stages and the tumour excised. The abdomen was closed with a polythene tube drain into the lesser sac. The possibility of a congenital cyst was considered, but excluded on the basis of the normal pancreatic tissue remaining and the appearance of the tumour.

POST-OPERATIVE COURSE.—Post-operative recovery was uneventful and within three weeks of the operation she said that she had "not felt so well for years". Four months after operation she continues in excellent health.

Investigations after operation showed no sign of pancreatic insufficiency; the urine never reduced Benedict's solution. A fat balance test showed that she absorbed 97 per cent of fat ingested. The anæmia responded to oral iron therapy and was of simple hypochromic iron-deficiency type.

PATHOLOGICAL REPORT.—The pancreatic tumour was a lobulated polycystic mass, 7 × 4 × 4 cm., some of the cysts containing clear fluid and some containing altered blood. Histological examination of sections made from side to side through the tumour showed a benign cystadenoma of the pancreas. No signs of malignant change were present. (*Figs.* 687, 688.)

COMMENT

Cystadenoma of the pancreas is most commonly found in middle-aged women. Of 28 cases reviewed by Benson and Gordon (1947) the average age was 48 years (17–76 years), and only 3 were male, 2 of these being the patients described by Carling and Hicks (1925). It is surprising that, apart from one Irish case (Pringle, 1925), no cases of pancreatic cystadenoma in female patients have been described in the British Isles. A cystadenoma of the pancreas typically produces few symptoms, and such symptoms as do occur are usually the result of pressure by the tumour on the surrounding structures (Herman and Asbel, 1949). The commonest symptom is a dull aching abdominal pain which is usually confined to the upper left quadrant (Neef, Rohdenburg, and Ritter, 1939; Benson and Gordon, 1947), or which may be generalized (Carter and Slattery, 1936). In the case described above abdominal pain was absent. There may also be pain in the back (Jemerin and Samuels, 1948), as there was in this case and as frequently occurs in other pancreatic conditions. Sometimes, however, there may be no pain but only a generalized feeling of fullness in the abdomen. Beloff (1945) and Herman and Asbel (1949) have described 2 cases in which this was the only symptom. Loss of weight is a rare symptom in this condition, Benson and Gordon (1947) being the only writers who record it in recent cases, whereas loss of weight and weakness were the commonest symptoms in cases of benign pancreatic cyst described by Judd, Mattson, and Mahorner (1931). Loss of appetite is also unusual and is presumably due to pressure on the stomach by the tumour. Vomiting may occur for the same reason. Constipation may be present (Neef and others, 1939; Bowers, Lord, and McSwain, 1942) probably owing to pressure on the colon by the tumour, for when it is removed the constipation ceases. A further non-specific symptom, which was prominent in the present case, is that of tiredness (Neef and others, 1939; Bowers and others, 1942). Occasionally the patient first discovers that she has something wrong by finding a lump in the abdomen (Bowers and others, 1942; Haukohl and Melamed, 1950). Associated with a cystadenoma there may be signs of pancreatic insufficiency. Of 28 cases reviewed by Benson and Gordon (1947) 6 had diabetes mellitus, which in one of these cases (described by Carling and Hicks, 1925) became more severe post-operatively, and which in one of the 5 cases described by Bowers and others (1942), became less severe after operative removal of the tumour. Loose fatty stools may also occur (Haukohl and Melamed, 1950).

The symptoms may be summarized as abdominal pain or fullness, pain in the back, tiredness, vomiting, constipation, loss of appetite, and loss of weight, which may be associated with signs of pancreatic insufficiency.

In spite of the rarity of this tumour, it should always be borne in mind as a possible diagnosis when a mobile abdominal mass is present, especially one in the upper abdomen. The diagnosis may sometimes be confirmed pre-operatively by a plain radiograph of the abdomen, when calcification in the tumour may be seen (Herman and Asbel, 1949; Haukohl and Melamed, 1950). A further occasional radiological sign is a displacement of the viscera by the tumour as revealed by a barium meal and enema (Bowers and others, 1942; Haukohl and Melamed, 1950). That such a tumour is rare is well shown by the fact that of 723,397 patients admitted to the Mayo clinic only 88 were proved to have a pancreatic cyst of any type (Judd and others, 1931). In a later paper Mahorner and Mattson (1931) showed that only 2 of these cysts were true cystadenomata.

This case demonstrates the fact that the best treatment is operative removal of the whole tumour, as other writers have shown.

I would like to thank Dr. H. S. Le Marquand (to whom I am also indebted for helpful suggestions) and Mr. Gordon Bohn for their permission to publish this case; and Dr. J. Mills for the histological report, and the University of Bristol, Pathology Department, for the photomicrographs.

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PRIMARY CARCINOMA OF THE URETER

By ANTHONY BARON and J. A. S. GREEN, London

PRIMARY carcinoma of the ureter has been generally regarded as a very rare disease, and received very little attention in surgical literature before the fourth decade of this century. In 1930, Rousselot and Lamon were able to trace records of only 49 cases, and they added 1 case of their own. Subsequent papers (Scott, 1934; Rusche and Bacon, 1938; Foord and Ferrier, 1939; Scott, 1943; Lazarus and Marks, 1945) raised the number of cases described

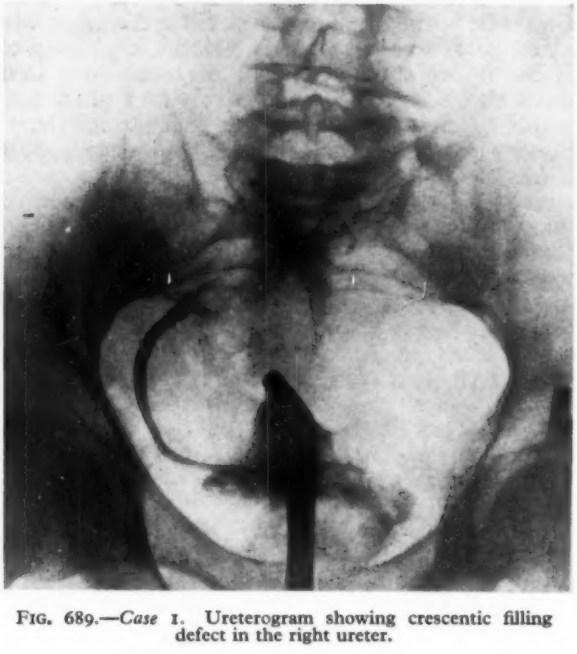

FIG. 689.—*Case* 1. Ureterogram showing crescentic filling defect in the right ureter.

to 183 by 1945. In August, 1951, Bowen and Atherton reported 2 new cases and Soloway 7 cases, and the total of case reports available for study was then 262. There has thus been a steady increase in the average number of cases reported each year, and it is becoming plain that the disease is much less rare than was earlier supposed. From the clinical point of view, the most interesting aspect of ureteric tumours is their diagnosis, and quite a brief aquaintance with them quickly teaches the ease with which they may be missed. It is our object in describing 11 cases of ureteric carcinoma to draw from personal experience certain conclusions about the clinical aspects of ureteric tumours, as well as briefly reviewing the literature.

CASE REPORTS

Case 1.—A man, aged 69, first attended hospital in August, 1951. Five weeks previously he had had an attack of right renal pain accompanied by retching, fever, and frequency of micturition. There had been no hæmaturia. His own doctor diagnosed pyelitis and treated him with sulphonamides. His condition improved, but he had further attacks of right-sided pain radiating to the testis.

Clinical examination revealed no abnormality. The urine was found to be heavily infected with *Bacillus coli*. An intravenous pyelogram showed a normal left kidney and ureter, but no function on the right side. Cystoscopy revealed no abnormality in the bladder, but when catheterization of the right ureter was performed the catheter was arrested at 10 cm. and a ureterogram showed the typical crescentic filling defect caused by the lower margin of a growth projecting into the ureter (*Fig.* 689).

At operation on Sept. 25, 1951, the tumour was found distending the ureter at the level of the bifurcation of the common iliac artery. It was noted that the ureter above the growth was not dilated. There were no apparent metastases and nephro-ureterectomy was performed. Convalescence was uneventful.

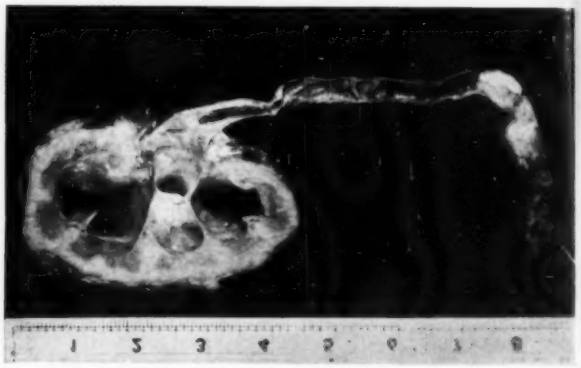

FIG. 690.—*Case* 1. The specimen, showing hydronephrotic kidney and the ureter occluded by solid growth.

Examination of the specimen revealed that the kidney was hydronephrotic and that the ureter was occluded by solid growth (*Fig.* 690).

Microscopical examination showed that the tumour was a solid transitional-cell carcinoma infiltrating the muscle coat.

The patient was last seen in July, 1952, when he was well with no evidence of recurrence.

Case 2.—A man, aged 44, first attended hospital in March, 1944, when he gave a history of terminal hæmaturia for three months.

Clinical examination was unrevealing. An intravenous pyelogram showed no abnormality on either side, but cystoscopy revealed an ulcerated papillary growth on the left lateral wall of the bladder, and this was destroyed by fulguration. He remained free of symptoms until 1946, when his hæmaturia recurred. The left ureteric orifice was now found to be obscured by an area of bullous œdema, which was fulgurated. Two years later the cystoscopic appearances were unchanged, but an intravenous pyelogram now showed some dilatation of the left renal pelvis though the lower end of the ureter was not clearly seen. In August, 1949, a biopsy was taken from the bladder and this showed mucosal hyperplasia only. Later in the year it was noticed that there were definite tubercles in the region of the left ureteric orifice. A catheter introduced into the left ureter was arrested at 2 cm. Ureterography was attempted, but the injection gave rise to considerable pain and the procedure was abandoned. Another biopsy was taken and the report was the same as on the previous occasion. The guinea-pig test was negative for tuberculosis. In February, 1951, an intravenous pyelogram showed that

the left kidney was now functionless, and on cystoscopy the left ureteric orifice was obscured by a hyperæmic area containing numerous millett-like tubercles. Soon afterwards the patient developed pain in the left loin, with fever. It was considered that he was suffering from a non-specific ulcer of the 'Fenwick' type and that the ureter had become obstructed by fibrosis.

On April 2, 1951, nephro-ureterectomy was performed, with removal of the affected area of the bladder.

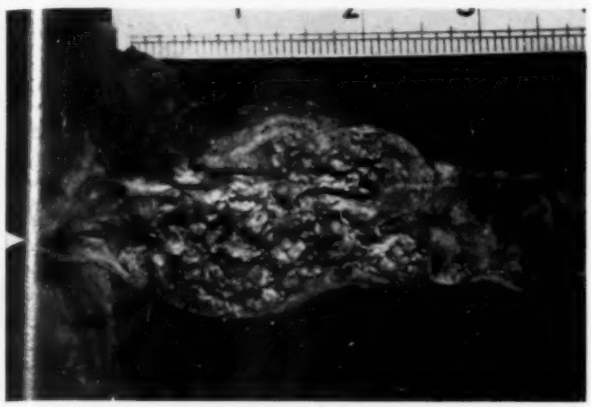

FIG. 691.—*Case* 2. The lower end of the ureter, with a cuff of bladder wall, showing the lumen largely filled with growth.

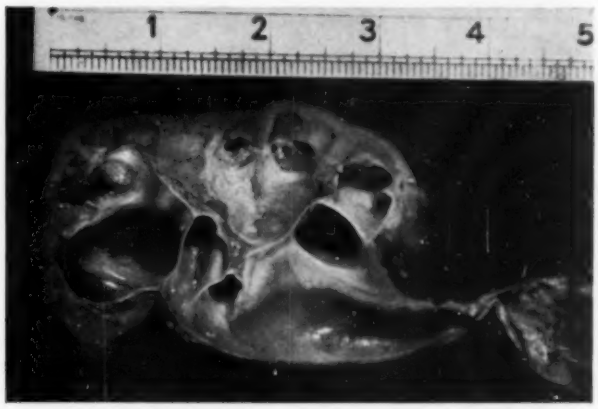

FIG. 692.—*Case* 2. Kidney and upper ureter showing the gross hydro-ureter and hydronephrosis.

Examination of the specimen showed a papillary transitional-cell carcinoma of the lower end of the ureter, infiltrating the muscle coat and largely filling the lumen (*Fig.* 691). The ureter above the growth was dilated and the kidney hydronephrotic (*Fig.* 692).

The patient made a satisfactory recovery from the operation, but died four months later from congestive cardiac failure due to mitral stenosis with auricular fibrillation.

Case 3.—A man, aged 42, first attended hospital in July, 1938, with a history of frequency of micturition for six months and of two attacks of hæmaturia.

On clinical examination the left kidney was palpable but no other abnormality was detected. An intravenous pyelogram showed a normal right kidney, but the left renal pelvis and calices were moderately dilated. Cystoscopy showed some basal cystitis. A ureteric catheter was passed for 30 cm. on the right side, but on the left side catheterization failed.

On July 22, 1938, exploration was undertaken. The kidney was hydronephrotic and the upper ureter dilated. A hard mass was palpated in the ureter 3 in. below the pelvi-ureteric junction. The kidney was removed, together with as much of the ureter as could be reached from the loin incision.

Examination of the specimen showed a yellow-white stricture in the ureter ½ in. in length.

Microscopically this was shown to be a carcinoma, arising from the villous papilloma.

The patient died from metastases in March, 1941.

Case 4.—A woman, aged 71, first attended hospital in May, 1939, and gave a history of frequency of micturition and a dragging pain in the left loin of six months' duration. She had had hæmaturia on three occasions.

On abdominal examination the left kidney was palpable and on pelvic examination a mass 2 in. in diameter was felt in the line of the left ureter. Intravenous pyelography showed no function on the left side. At cystoscopy the left ureteric orifice was seen to be drawn up and everted. On the right side a ureteric catheter was passed for 28 cm., but on the left it was held up at 10 cm. A satisfactory ureterogram was not obtained.

Exploration was carried out a week later through a lumbar incision, and the left kidney, pelvis, and ureter were found to be dilated. The ureter was traced to the pelvic brim, where a hard, adherent mass was noticed, and during the manipulation the ureter was torn across at the upper level of this mass. The lower end of the ureter was then explored through a left inguinal incision, but it was found impossible to remove the mass in this region.

Examination of the kidney and attached portion of the ureter showed marked hydronephrosis. The ureter at the lower end of the specimen was infiltrated by a transitional-cell carcinoma spreading in the submucosa. The growth was also present in a small vein.

This patient died in August, 1940. No post-mortem examination was carried out.

Case 5.—A woman, aged 70, first attended hospital in October, 1948, with a history of pain in the left loin for eighteen months. She also suffered from nocturnal frequency of micturition and had had several slight attacks of hæmaturia.

On examination the left kidney was palpable and tender. The urine contained pus and red cells and on culture grew *Bacillus coli*. An intravenous pyelogram showed a normal right kidney and ureter, but there was no function on the left side (*Fig.* 693). On cystoscopy the bladder was normal. The left ureter admitted a catheter to 6 cm. only and ureterography revealed a block in the ureter at the middle of the third spindle (*Fig.* 694). It was not appreciated that this was due to growth, and operation was decided upon to remove a painful, functionless kidney associated with a stricture of the ureter.

The left kidney was explored on Jan. 8, 1949, and found to be hydronephrotic. The ureter was followed downwards, but broke off somewhere below the pelvic brim. The operation was then terminated as the patient's general condition gave rise to anxiety. On Feb. 11 the stump of the ureter was removed through a midline subumbilical incision, together with an enlarged gland in the neighbourhood.

Examination of the specimen showed a grossly hydronephrotic kidney and the ureter occluded by growth.

Microscopically this was shown to be a transitional-cell carcinoma. The gland removed was not invaded.

The patient's subsequent progress was satisfactory until nine months later, when she developed pain in the back and a radiograph on Dec. 2, showed collapse of the

first lumbar vertebra which was thought to be due to a secondary deposit (*Fig.* 695).

Deep X-ray therapy was then instituted. Despite continuing pain in her back she has nevertheless remained well in other respects, and was last seen on Sept. 3, 1952, when radiography showed collapse of the first four lumbar vertebræ. The diagnosis was then revised to one of senile osteoporotic collapse.

FIG. 693.—*Case* 5. Intravenous pyelogram. Note absence of secretion on the left side.

FIG. 695.—*Case* 5. Plain radiograph of spine showing collapse of the first lumbar vertebra.

Case 6.—A woman, aged 48. In June, 1948, this patient underwent a hysterectomy for "very early" carcinoma of the body of the uterus. In Decembe the same year she experienced left-sided renal colic, which was followed by the passage of "a piece of flesh" and later by slight hæmaturia.

Clinical examination revealed no abnormality. The urine contained red blood-cells and was sterile on cu ure.

FIG. 694.—*Case* 5. Ureterogram showing block and filling defect in the left ureter.

An intravenous pyelogram showed a marked hydronephrosis on the left side (*Fig.* 696). On Feb. 4, 1949, cystoscopy was carried out. The bladder was normal and a ureteric catheter passed readily to 25 cm. on the left side, but a satisfactory retrograde pyelogram was unfortunately not obtained. On July 26, she had another severe attack of renal colic and passed more tissue which she gave to her doctor, who had it microscopically examined. The report on this fragment was transitional-cell carcinoma. The left ureter was again catheterized and this time the catheter was arrested at the level of the pelvic brim. Ureterography now demonstrated a definite filling defect of the ureter in the region of the fifth lumbar vertebra (*Fig.* 697).

On Aug. 5, the kidney and ureter were explored. The kidney was hydronephrotic and a fusiform swelling was found in the ureter at the junction of the middle and lower thirds.

Nephro-ureterectomy was performed.

Examination of the specimen showed gross hydronephrosis and a papillary tumour growing into the lumen of the lower end of the ureter. Histologically this tumour was a papillary transitional-cell carcinoma which did not invade the muscle coat. On Aug. 13, 1951, the patient was found to have a hard mass palpable in the left side of the pelvis, but whether this was secondary to the ureteric or the uterine growth is undecided. She was given a course of radiotherapy and was last seen on July 30, 1952, when she was in some pain and the mass was still palpable on pelvic examination.

Case 7.—A man, aged 53, first attended hospital in 1940 with a history that for two weeks he had had a painless hæmaturia with the passage of clots and also a dull intermittent pain in the left testicle. Two years

previously he had had a similar attack, which had been diagnosed as pyelitis.

Clinical examination revealed a greatly enlarged left kidney which intravenous pyelography showed to be functionless. On cystoscopy the bladder was normal, and the ureteric catheter was arrested at 10 cm. on the left side. No retrograde pyelogram was, however, obtained.

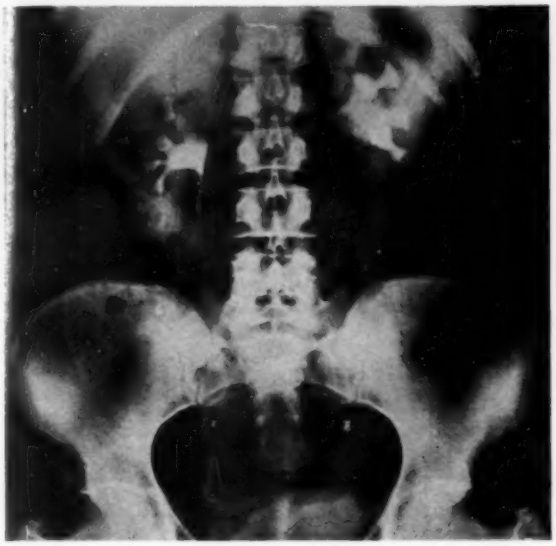

FIG. 696.—*Case* 6. Intravenous pyelogram showing marked hydronephrosis on the left side.

On May 7, 1940, exploration was carried out and the left kidney was found to be hydronephrotic. The ureter was dilated and a lump the size of a walnut was present in it at the level of the pelvic brim. The ureter was divided well below this and the kidney and ureter removed.

The histological report on the ureteric tumour was papilloma.

On Feb. 19, 1946, the patient had a further attack of severe painless hæmaturia. On cystoscopy a large papilloma was seen in the region of the left ureteric orifice, which was fulgurated and later removed by open operation through the bladder. Histologically this growth showed "early malignancy". The patient did not attend again until July 29, 1949, following a further attack of hæmaturia. On cystoscopy a large villous growth was seen on the left side of the bladder.

On Oct. 7, the ureteric stump was removed together with the adjacent part of the bladder and found to be distended by a tubular mass of growth. The histological report on this specimen was papillary transitional-cell carcinoma of a low-grade malignancy. He was next seen on May 4, 1951, with a history of five days' hæmaturia. Cystoscopy now revealed two nodular tumours near the vault of the bladder.

On July 6 these two tumours were removed by partial cystectomy and the histological report was "transitional-cell carcinoma of low-grade malignancy. Invasion of the bladder wall is not marked and the lines of incision pass well clear of the growth".

The patient attended again in November and was found to have diffuse recurrence of the growth, which had invaded the peritoneum and was adherent to a loop of small intestine. Total cystectomy was carried out on Nov. 27, but the patient died on Dec. 5.

At post-mortem examination some of the mesenteric lymphatic glands were found to contain secondary deposits.

Case 8.—A man, aged 48, first attended hospital on March 31, 1952, with a history of two attacks of hæmaturia and of pain in the left groin for three months.

Clinical examination revealed no abnormality. On intravenous pyelography the right kidney and ureter appeared normal, but there was no excretion of dye on the left side. Cystoscopy showed a normal bladder, and on catheterization of the left ureter the catheter was arrested at 11 cm. Retrograde urography revealed

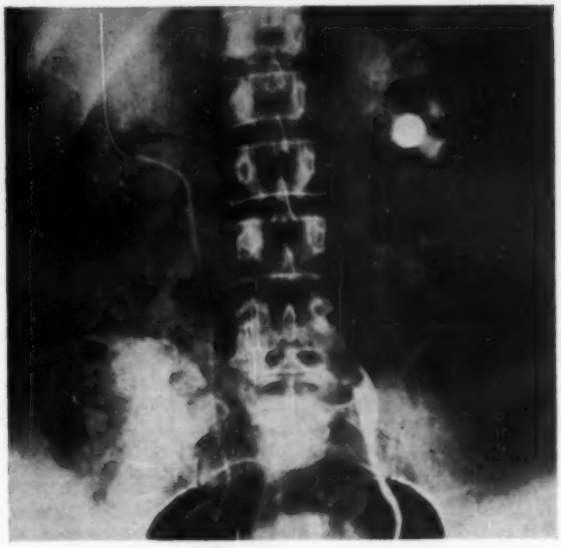

FIG. 697.—*Case* 6. Ureterogram showing filling defect of the left ureter in the region of the fifth lumbar vertebra.

FIG. 698.—*Case* 8. Retrograde ureterogram showing filling defect due to the ureteric growth on the left side. The opaque medium has passed the point of obstruction and outlines a greatly dilated ureter and renal pelvis.

the typical filling defect of a ureteric growth. The ureter above the growth was dilated and the kidney hydronephrotic (*Fig.* 698).

On May 12 the left kidney and ureter were removed. The ureteric growth was mobile and situated at the pelvic brim, and no enlarged glands were evident. Convalescence was uneventful.

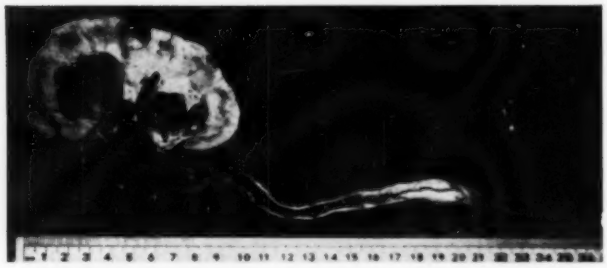

FIG. 699.—*Case* 8. Left kidney and ureter removed at operation, showing the tumour obstructing the ureter and the marked back-pressure changes.

Examination of the specimen showed a tumour 2 in. in length obstructing the ureter and producing marked back-pressure changes (*Fig.* 699). Histologically the growth was a papillary transitional-cell carcinoma invading the muscle coat but not the adventitia (*Fig.* 700).

FIG. 701.—*Case* 9. Stump of the ureter removed at second operation and filled with solid growth.

Case 9.—A woman, aged 71, first attended hospital in 1948 complaining of hæmaturia and pain in the back for three months.

Clinical examination was unrevealing, but intravenous pyelography showed a hydronephrosis on the right side without dilatation of the upper ureter. A review of these films showed a filling defect in the pelvic portion of the ureter, but this was not noted at the time.

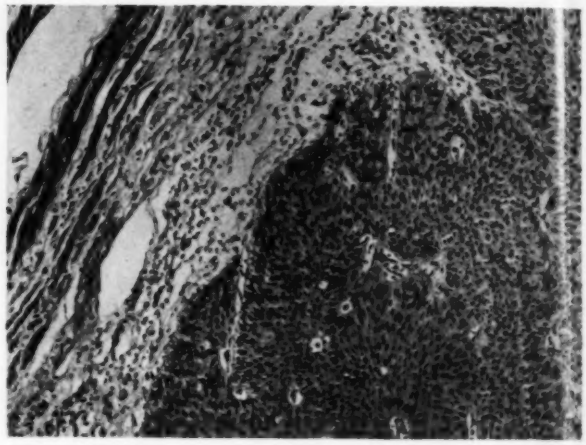

FIG. 700.—*Case* 8. Microscopical section of the tumour. Papillary transitional-cell carcinoma invading the muscle coat. (× 228.)

Retrograde pyelography was performed, the catheter passing readily to the pelvis.

A fortnight later the right kidney was removed and the specimen showed inflammatory changes, atrophy, and fibrosis, but no other pathology. After the operation she had an intermittent blood-stained discharge from the nephrectomy sinus, pain in the right loin, and occasional hæmaturia. In February, 1951, cystoscopy was performed and a discharge of blood was seen coming from the right ureter. A ureterogram now showed a filling defect in the ureter. Unfortunately both these films and those of the original intravenous pyelogram in 1948 were lost before we were able to get the slides made.

A further operation was carried out in February, 1952, and the ureteric stump was found to contain growth adherent to the peritoneum but not to the iliac vessels. The stump, together with an enlarged gland, was removed.

The specimen was found to contain a solid growth (*Fig.* 701). Histologically this was a transitional-cell carcinoma. The lymph-gland was extensively involved.

In March the patient complained of pain in the left thigh and a radiograph showed an osteolytic metastasis in the upper third of the left femur. In April a pathological fracture occurred and she went downhill and died about a month later.

Post-mortem examination showed secondary deposits in the para-aortic lymph-nodes, the porta hepatis, in both lobes of the liver and in the left lung, in addition to the metastasis in the femur.

Case 10.—A man, aged 77, and blind from glaucoma, who had the following history :—

In 1934 he had undergone right nephrectomy for hæmaturia. In 1937 he had had papillomata of the bladder fulgurated. In 1946 he underwent prostatectomy for acute retention of urine. This was followed by further fulgurations in 1947, but he later developed repeated attacks of hæmaturia. In 1949 cystoscopy showed a raised solid carcinoma covering the right side of the trigone and a sausage-shaped mass could be felt bimanually in the line of the right ureter. A biopsy taken from the bladder showed a low-grade, papillary, transitional-cell carcinoma.

Owing to his age it was decided to treat the patient with palliative fulgurations and these have been carried out every two or three months with relief of symptoms. He was last seen at the end of 1951, when there was no significant change in his condition.

DISCUSSION

Recent writers (Larazus and Marks, 1945; Mortensen and Murphy, 1950) have stressed that this condition is much commoner than is generally supposed. Our personal experience strongly supports this view, and it is probable that the apparent rarity of the disease is due to the difficulty of diagnosis and to the fact that the symptoms can nearly always be explained by associated changes found in the renal tract. The only reported cases are, therefore, those which are operated upon or which die in hospital and are submitted to autopsy. Many patients are undoubtedly missed and die at home with some other diagnosis.

Age Incidence.—In previously reported cases the condition has been most commonly seen in the sixth and seventh decade. The youngest sufferer was aged 22 years and the oldest 89. Of the patients, 66 per cent were males and the lower end of the ureter was the site of growth in half the cases. In this small series there were 7 men and 4 women. The oldest patient was 77 years of age and the youngest 42. Six of the eleven tumours were situated at the level of the pelvic brim or below.

Clinical Features.—The leading symptoms are hæmaturia, pain, and tumour (Scott, 1943).

Hæmaturia occurred in 70 per cent of reported cases and is stated to have been very variable in degree and sometimes accompanied by colic. In the cases here reported it was the leading symptom in 4 (*Cases* 2, 7, 8, and 10), was present together with pain or frequency in 4 (*Cases* 3, 4, 5, and 9), was very slight in 1 (*Case* 6), and absent in the remaining 1 (*Case* 1). In 5 cases (*Cases* 3, 4, 5, 6, and 9) the history of bleeding was only elicited on questioning, as it was both slight and infrequent.

Pain is reported in 64 per cent of published cases and stated to be either obstructive renal pain, renal colic, or pain from metastatic involvement. We have encountered each of these three forms, and in addition two of our patients complained of pain in the corresponding testicle and groin, intermittent and unassociated with attacks of colic.

Tumour or swelling is reported in 40 per cent of published cases. The usual finding has been a palpable hydronephrotic kidney, but on rare occasions the growth itself has been felt on pelvic examination. A palpable swelling in the loin was present in 4 of our cases and in 2 cases the ureteric growth was felt on bimanual examination (*Cases* 4 and 10).

In *Case* 1 the onset of symptoms was associated with high fever, and a diagnosis of pyelitis was confidently made by the practitioner in charge.

INVESTIGATIONS

The Urine.—This may be normal or may contain red cells or frank blood. Fragments of growth may be passed, and this occurred in *Case* 6 of this series. In *Case* 1 the urine was heavily infected with *B. coli*.

Plain Radiograph.—This may show an enlarged renal shadow on one side. It may also show stones—a complication not encountered in this series. Stones are, of course, not uncommonly associated with malignant disease of the urinary tract, notably in the renal pelvis and bladder.

Intravenous Pyelography.—Scott (1943) states that "in the diagnosis of this condition intravenous pyelography is unhelpful". This is true in so far as it is rare for the lesion itself to be seen on an intravenous pyelogram, though this can happen, and in fact did so in *Case* 9 of our series. (Unfortunately it was not recognized, for though a hydronephrosis was present there was no dilatation of the ureter above the growth. A ureter of normal calibre was also present in *Case* 1.) Quite apart from such direct help, intravenous pyelography is of value because the findings in these cases are constant, and in any event it is so frequently carried out at an early stage of a urological examination that it is most important to be familiar with the common findings in carcinoma of the ureter. The most frequent occurrence in this series was a non-functioning kidney on the side of the tumour (*Cases* 1, 2, 4, 5, 7, 8, and 11), and in the remaining cases hydronephrosis was present. It is important to remember that despite the presence of hydronephrosis there may be no dilatation of the ureter (*Cases* 1 and 9), especially in view of the opinion expressed by Masina (1950), writing on tumours of the vesical end of the ureter, who states that "the common syndrome in these cases is the presence of a dilated ureter on an intravenous pyelogram associated with a bladder lesion which is not palpable as an infiltrating lesion on bimanual examination. As soon as these two findings are encountered a tumour of the terminal part of the ureter should be suspected". Certainly, with lesions in the region of the brim of the pelvis, hydro-ureter may be absent.

Retrograde Pyelography.—It is generally agreed that this is the most valuable investigation in ureteric carcinoma, because it may demonstrate a block by obstruction to the passage of the catheter or because this passage may provoke bleeding. Further, ureterography should show a characteristic crescentic filling defect (*Figs.* 689, 694, and 697). There are, however, several difficulties and sources of error, both in technique and interpretation, which merit further consideration.

1. The catheter may pass the site of a ureteric lesion and reach the renal pelvis without difficulty and without provoking bleeding (*Cases* 6 and 9). This is, of course, particularly likely to occur when the growth is small, and it is most important that this finding should not produce a false sense of security and lead to only half-hearted efforts being made to obtain good pictures of the pelvis and particularly of the ureter.

2. On rare occasions it may be impossible to introduce the catheter (*Case* 3). Usually, however, a second or third attempt is successful, and it is of great importance that repeated attempts should be made especially if treatment on conservative lines is being considered.

3. The ureteric catheter may be passed to a certain distance and then held up, but a subsequent ureterogram fail to show a filling defect. If this happens it

is very likely that a diagnosis of 'stricture' will be made and the possibility of carcinoma overlooked (*Cases* 2, 5, and 7). This occurrence can, we believe, be avoided by attention to the following points in technique: First, the examination should not, if it can possibly be avoided, be made under general anæsthesia. The catheter can usually be passed only for a short distance, and it readily becomes displaced if it is necessary to wait for the patient to become conscious or if he has to be transported from the cystoscopy theatre to the X-ray department. Furthermore, the films should be exposed while the injection is being made otherwise the ureter may not be sufficiently filled to demonstrate a filling defect.

Failure to demonstrate the filling defect at the first examination may have some unfortunate consequences. Thus, in *Case* 6 five months elapsed before the diagnosis was made and in *Cases* 2 and 5 operation was only carried out after considerable delay because of persistent pain in the functionless kidney. Exploration itself is not an infallible method of detecting ureteric carcinoma if it is not specifically looked for. In *Case* 9, and possibly *Case* 10 also, the growth was missed during performance of nephrectomy.

To obtain a clear picture of the whole ureter, or at least of the ureter up to the point of obstruction, is thus of vital importance in diagnosis. Further, we would suggest that 'stricture' of the ureter at any rate in its central portions should be regarded as malignant until proved otherwise.

DIAGNOSIS

Lazarus and Marks (1945) state that a correct pre-operative diagnosis has been arrived at in 39 per cent of reported cases. This includes their own 3 cases, all of which presented with hæmaturia. In the 11 cases here reported, a correct pre-operative diagnosis was made in 5, but in 3 of them under unsatisfactory conditions. In *Cases* 4 and 10 the growth was sufficiently advanced to be palpable and the latter patient had also had a nephrectomy on the same side some years before. In *Case* 6 the diagnosis, although correctly made, was fortuitous and delayed. In only 2 patients (*Cases* 1 and 8) was the condition conclusively demonstrated at an early stage by a good ureterogram.

Though, as we have said, exploration of the kidney does not invariably lead to the discovery of a ureteric growth, it does so sufficiently often to make it a fortunate circumstance that renal symptoms usually prompt exploration sooner or later. A consideration of the reason why this was undertaken in our cases gives a good idea of the differential diagnosis of this disease. In *Cases* 2 and 5 surgery was carried out to remove a painful, functionless kidney associated with a 'stricture' of the ureter. In *Case* 7 the profuse hæmaturia and grossly enlarged functionless kidney suggested a diagnosis of hypernephroma. In *Case* 6 the original diagnosis was idiopathic hydronephrosis, and only the passage of a piece of growth—fortunately preserved—led to an alteration of this. In *Case* 9 nephrectomy was also carried out for painful hydronephrosis, though in this case the ureteric growth was missed and gave rise to a blood-stained discharge from the incision, for more than three years afterwards. In *Case* 2 a diagnosis was made of Fenwick's ulcer associated with a stricture of the ureter and hydronephrosis.

It is, therefore, evident that primary ureteric carcinoma may readily elude diagnosis despite the carrying out of what is usually regarded as a complete urological investigation, even including exploration of the kidney. Though there are numerous sources of difficulty which have already been discussed, there are two outstanding reasons why this condition is so frequently missed. First, it is not thought of, and secondly sufficiently determined efforts are not made to obtain adequate pictures of the whole urinary tract. The diagnosis is not really so difficult once the condition is suspected. There are two syndromes, one merely representing a later stage than the other, which may be recognized if clinical and pyelographic findings are considered together.

1. Hydronephrosis, usually associated with hæmaturia, but not necessarily associated with hydroureter. Ureteric catheterization may fail to demonstrate a block.

2. The commoner syndrome of a functionless kidney usually with a history of hæmaturia and with some obstruction demonstrable in the ureter.

When one of these sets of conditions is encountered, it is essential to exclude ureteric carcinoma, and the only really reliable method of doing this is by ureterography properly carried out. In the case of the first 'syndrome', this amounts to a plea for thorough investigation of any hydronephrosis. Too much emphasis is apt to be placed upon the value of obtaining good pictures of the renal pelvis to the detriment or neglect of the ureter. The investigation of the urinary tract by retrograde radiography should not be thought of as 'retrograde pyelography' but as 'uretero-pyelography' or urography. Only thus will the ureter receive the attention it deserves.

Another lesson learned from the cases here reported is the danger of attributing back-pressure changes producing a functioning or non-functioning hydronephrosis to a 'stricture' of the ureter of unspecified but benign aetiology. This was done three times and each time the stricture turned out to be malignant. The diagnosis of stricture of the ureter, except at its two ends, should not be accepted until exhaustive efforts have been made to exclude carcinoma.

We feel that if the diagnosis of ureteric carcinoma is to be made more frequently at an early stage, the following points are worth bearing in mind:—

1. Carcinoma of the ureter is not a very uncommon disease.

2. A urological investigation which does not include adequate visualization of both ureters is not a full urological investigation.

3. Stricture of the ureter associated with hydronephrosis, functioning or non-functioning, should be regarded as malignant until proved otherwise.

4. Hydronephrosis in a patient of the cancer age is a diagnosis which demands elaboration, especially in the presence of hæmaturia.

PATHOLOGY

Growths of the ureter arise from epithelium similar to that of the bladder and may be classified in a manner identical with that adopted by Dukes

and Masina (1949) for growths of that organ. Lazarus and Marks (1945) adopt a less detailed classification of ureteric growths into three main groups: (*a*) Papillomata; (*b*) Papillary carcinomata; (*c*) Non-papillary carcinomata. Papillary growths are frequently multiple and solid growths single, but the two may coexist, as in the following case, which we have not included among the rest as there is some doubt whether the ureteric growth was the primary one. It seems likely that it was an instance of multicentric origin of cancer rather than an instance of 'seeding', since the growth in the renal pelvis was small in comparison to that in the ureter.

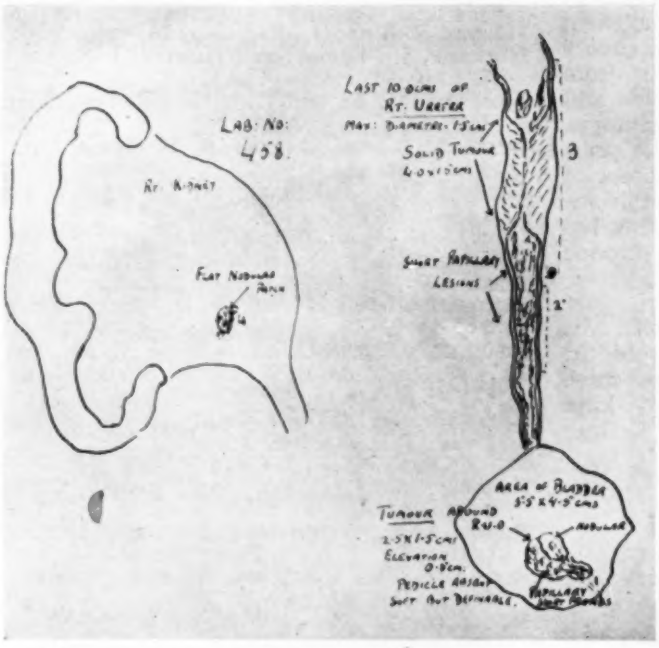

FIG. 702.—*Case* 11. Schematic drawing of the specimen (*Fig.* 703) and of the renal pelvis.

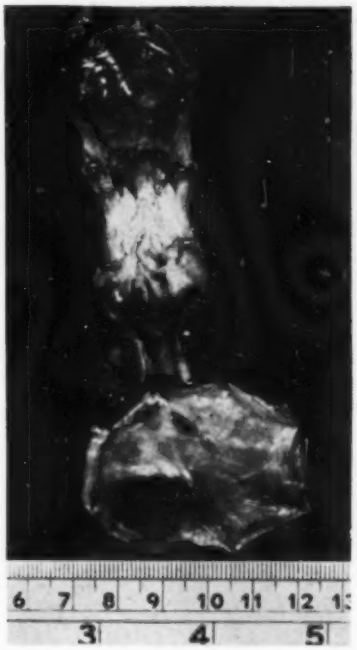

FIG. 703.—*Case* 11. The lower end of the ureter and the cuff of bladder removed at operation.

Case 11.—A man, aged 44, first attended hospital on July 6, 1949, with a history of hæmaturia for two days. There were no abnormal findings on clinical examination. At cystoscopy a solid tumour was seen at the right ureteric orifice. Intravenous pyelography showed that there was no function on the right side. Catheterization of the right ureter was not possible and nephro-ureterectomy and partial cystectomy were carried out.

Examination of the specimen revealed a solid carcinoma, 4 × 1·5 cm., in the ureter at the junction of the lower and middle thirds, with small papillary lesions below this. At the ureteric orifice there was a mixed nodular and papillary growth. The ureter above the solid growth was dilated, and marked hydronephrosis was present, with a flat nodular patch in the renal pelvis (*Figs.* 702, 703).

This patient was last seen two years after the operation, when he was alive and well with no evidence of recurrence.

TREATMENT

The treatment of tumours of the vesical end of the ureter has been fully dealt with by Masina (1950). Tumours elsewhere in the ureter are best treated by primary nephro-ureterectomy, and the deliberate adoption of this method depends upon a correct pre-operative diagnosis. In view of the not infrequent recurrence of growth in the ureteric stump if one is left (*Cases* 7 and 10), it seems advisable at the primary operation to remove the whole ureter together with a small cuff of bladder around the ureteric orifice. MacAlpine (1947) considered it safest, in dealing with cases of papillomatosis, to leave the lower end of the ureter in situ and subsequently to destroy its mucosa by cystoscopic diathermy. The danger of implanting malignant cells in the wound can, however, be almost completely avoided if the bladder is adequately mobilized so that it can be drawn into a cone at the site of the ureteric insertion before being clamped and divided. Although this is the ideal procedure, most growths fortunately are single in the first instance, and if the patient's condition becomes alarming the operation may be terminated after dividing the ureter below the obvious growth without much danger in the average case. Nevertheless, it is essential that such ureter as has had to be left behind should be dealt with at the earliest possible moment. The conservative removal of apparently benign tumours described by Vest (1945) seems to us to have very few indications and great disadvantages: The kidney is usually too damaged to be worth saving; there is a theoretical danger of implanting neoplastic cells in the wound; and the hazard of recurrence or further spontaneous neoplastic change in the renal tract is ever present.

It is well to bear in mind that if, as is usual, the kidney is approached first at operation, it is quite easy if the ureter is infiltrated with growth to tear it through at the site of the tumour whilst clearing it, especially in the region of the pelvic brim. This occurred in 2 cases in this series (*Cases* 4 and 5), both

of whom had advanced growths. It does not appear that the prognosis was affected in these cases by the accident, but it is clearly highly undesirable.

PROGNOSIS

The prognosis in carcinoma of the ureter is usually considered to be poor, though there are no figures available of any adequate series followed up for a reasonable time. No definite conclusions can be drawn from the small series reported here, but it is clear that, like growths in the bladder, ureteric neoplasms may be comparatively benign papillary lesions or infiltrating lesions of a more malignant nature. Thus *Case* 2 lived for seven years, *Case* 7 for eleven years, *Case* 10 for seventeen years from the date of onset of hæmaturia. *Case* 5 was still alive and well four years after the onset of symptoms. On the other hand, *Case* 3 was dead in three years and *Cases* 4 and 9 in one year from the onset of the first symptom. In *Case* 6 the position is indeterminate, as she has lived for four years, but has a recurrence which may be either ureteric or uterine in origin.

Our thanks are due to Mr. M. F. Nicholls, Mr. W. H. C. Romanis, and to the Surgeons of St. Peter's Hospital for permission to publish these cases. *Figs.* 702, 703 are reproduced by kind permission of Mr. F. Masina and the Royal College of Surgeons.

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THE PATHOLOGY OF GASTRIC ARTERIES, WITH SPECIAL REFERENCE TO FATAL HÆMORRHAGE FROM PEPTIC ULCER

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For at least one hundred years it has been believed that arteriosclerosis has an important relationship to peptic ulcer, and especially to fatal hæmorrhage from these ulcers. "It is our opinion that hypertension is an important cause of massive fatal hæmorrhage, and that its association is often difficult to diagnose clinically because the patient when seen is often in a state of extreme shock." (Anderson, 1948.) "A century ago it was suggested that only a vascular factor could account for the localized lesion of peptic ulcer. . . . There seems no reason why thrombosis should not occur in *end arteries* of the stomach in human subjects with arteriosclerosis, and this would account for the ulceration seen in elderly patients with no previous history whatever of an ulcer dyspepsia. Characteristic of this type of ulceration is the sudden onset of symptoms, often dated to a day, and the rapid healing that follows. Bürger (1947) has drawn attention to the striking parallelism between the incidence and growing frequency of angina pectoris and peptic ulcer in cases seen in Leipzig between 1931 and 1941, and interprets this as showing that both diseases have a common cause." (Avery Jones, 1952.) However, in the same book we read: "The freely anastomotic nature of the vessels of the stomach wall is perhaps its most notable feature. There are *no anatomical end arteries*."

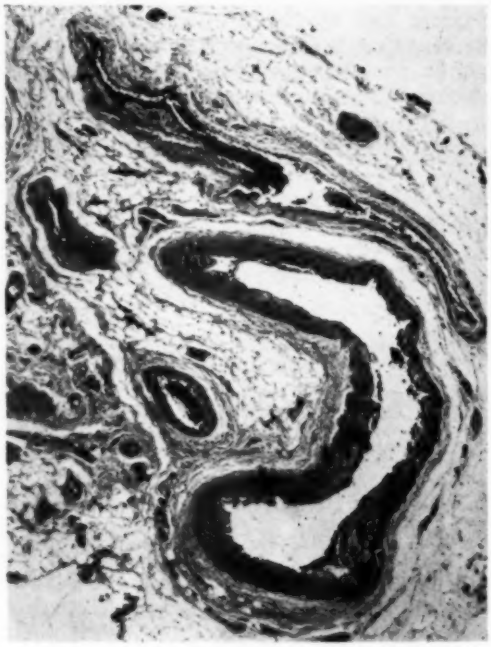

FIG. 704.—Normal gastric arteries from a woman, aged 80, dying of coronary thrombosis. In 50 such deaths the gastric arteries were normal in 45 and showed trivial lesions in 5. (× 11.)

FIG. 705.—Atheromatous plaque in a woman, aged 74, dying of coronary thrombosis; except for this her gastric arteries were normal. In 50 such cases a similar plaque was found in a man, aged 63; 3 had localized inflammatory arterial lesions and the remainder were normal. (× 11.)

Partial gastrectomy is now a common operation; 327 were performed in the Derbyshire Royal Infirmary in 1951. If arteriosclerosis in its typical forms (Monckeberg's sclerosis and atherosclerosis) is an important cause of peptic ulcer it should be common in stomachs removed surgically. It was not seen in any of the 1951 series, but has been seen in an early stage in one removed during 1952. Arterial lesions related to the ulcer do occur fairly regularly, but they are not 'arteriosclerosis'. The obvious normality of the blood-vessels in almost any stomach exposed at operation prompted a study of their condition in arteriosclerotic deaths, and for this purpose fatal cases of coronary thrombosis were examined.

THE GASTRIC ARTERIES IN DEATHS FROM CORONARY THROMBOSIS

Fifty consecutive deaths from coronary thrombosis were composed of 40 males aged 32–85 (average 62 years), and 10 females aged 55–93 (average 74 years) (*Table I*). The region of the lesser curvature with lesser omentum attached was taken for histological study together with the affected coronary artery. Because the gastric vessels all appeared normal macroscopically it was not possible to select the worst parts, for this reason from two to four likely blocks were taken from each. In 45

of the 50 cases the gastric arteries appeared totally normal (*Fig.* 704). In 3 (a man of 67, and two women both 79) small arterial lesions were found which were almost certainly of inflammatory origin, these were quite localized, the arteries generally being normal. Only two showed a lesion which could be called 'arteriosclerosis'. This was an atheromatous plaque too small to reduce appreciably the size of the lumen, all the other arteries appeared normal. The subjects were a woman of 74 and a man of 63 (*Fig.* 705).

Table I.—Gastric Arteries in 50 Consecutive Deaths from Coronary Thrombosis

40 males aged 32–85 (average 62 years)
10 females aged 55–93 (average 74 years)

A Few Gastric Arterial Lesions found in 5 Cases only :—
1. Female, 79. Evidence of recent gastric ulcer.
2. Male, 67. To gastric lymph-gland with dust reaction.
3. Female, 79. Sinus catarrh in gastric lymph-glands.
4. Female, 74. Small atheromatous plaque, large lumen.
5. Male, 63. Small atheromatous plaque, large lumen.

Some of the many coronary deaths with normal gastric arteries were instructive. A man aged 64 had a perforated duodenal ulcer in addition to complete occlusion of the main descending branch of the left coronary artery. He had much sclerosis in the splenic and superior mesenteric arteries, but all the gastric arteries were normal. There was no disease of any vessel near the perforated ulcer, though this was examined in one hundred and sixty sections. Perforation of a peptic ulcer is presumably a rapid process; this normality may therefore be correlated with later observations on acute erosion of arteries by the ulcer process. Another man aged 69 had a typical chronic duodenal ulcer, but all the gastric arteries were normal.

If the typical forms of arteriosclerosis cannot be accepted as having any relationship to peptic ulcer or massive hæmorrhage from these, hypertension may be of consequence. This problem was studied in the 26 fatal cases, the most important point being the weight of the heart. In one, a woman of 57, it was actually small, weighing only 6 oz.; in 14 it was normal in size and free from vascular and valvular disease; in 8 it was hypertrophied (weight in oz.: 13, 15, 15, 15, 16, 17, 20, 26). No large heart had advanced coronary disease; the largest had aortic regurgitation. Over two-thirds of the cases were thus almost certainly free from hypertension at the time the massive hæmorrhage developed. Although it is impossible to accept any aetiological relationship to the massive hæmorrhage from the peptic ulcer, it would seem reasonable

Table II.—Details of Twenty-six Deaths from Hæmatemesis (with Post Mortem), 1948–1952

A. Primary Hæmorrhage

Case No.	Sex	Age	Ulcer	Heart	Days	R.B.C.	Blood-urea	Treatment
1	M.	34	D	15 oz.	4	2·6; 1·7 m.	26	5 pt. blood
2	M.	49	G	N	1¼	2·9 m.	65	5 pt. blood
3	F.	54	G	20 oz.	0¾	3·6 m.	47; 114	1 pt. blood
4	F.	60	G	N	3	3·2; 4·0 m.	?	2 pt. blood
5	M.	65	D	N	14	2·3; 1·8 m.	40; 230	10 pt. blood
6	F.	66	G	13 oz.	2	2·9 m.	138	Gastrectomy
7	F.	66	G	N	2	2·2 m.	98	4 pt. blood
8	F.	75	G	N	3	4·2 m.	47	Gastrectomy

B. Primary Hæmorrhage while under Treatment

Case No.	Sex	Age	Ulcer	Heart	Days	R.B.C.	Blood-urea	Treatment
1	M.	57	D	N	0¼ (2)	?	?	None
2	M.	59	D	N	2 (4)	?	?	10 pt. blood
3	M.	77	O+D	15 oz.	6 (21)	2·6 m.	?	4 pt. blood
4	M.	89	G	16 oz.	1 (15)	?	?	None

C. Recurrent Hæmorrhage

Case No.	Sex	Age	Ulcer	Heart	Days	R.B.C.	Blood-urea	Treatment
1	M.	46	G (HG)	N	6+8	1·9 m.	?	17 pt. blood
2	M.	58	G	26 oz.	3+1	3·6 m.	?	3 pt. blood D.O.T.
3	F.	70	G	N	7+5	2·8 m.	?	2 pt. blood
4	F.	77	G	N	7+2	?	?	4 pt. blood

D. Hæmorrhage after Gastrectomy

Case No.	Sex	Age	Ulcer	Heart	Days	R.B.C.	Blood-urea	Treatment
1	M.	60	D	N	2	?	?	6 pt. blood
2	M.	67	D	15 oz.	2	?	?	5 pt. blood

E. Hæmorrhage plus Other Disease

Case No.	Sex	Age	Ulcer	Heart	Days	R.B.C.	Blood-urea	Treatment
1	M.	45	G	N	1	?	?	(Carcinoma of lung)
2	F.	57	G	6 oz.	? 1	?	?	(Hodgkin's)

F. Uræmia and Bronchopneumonia

Case No.	Sex	Age	Ulcer	Heart	Days	R.B.C.	Blood-urea	Treatment
1	M.	35	D	N	? 7	?	50	None (tramp)
2	M.	41	D	N	8	4·4 m.	272	Gastrectomy
3	M.	47	D	N	5	?	280	Gastrectomy
4	M.	66	2 D	17 oz.	4	3·9 m.	240	5 pt. blood
5	M.	68	D	N	8+8	1·3 m.	195	3 pt. blood, plus 3 pt. blood
6	F.	77	G	N	2, 5, 2, 5	4·0 m.	300	3 pt. blood, plus 1 pt. blood

D, Duodenal ulcer; G, Gastric ulcer; HG, Hour-glass stomach; O, Œsophageal ulcer; N, Heart of normal weight with normal arteries and valves; D.O.T., Died on operating table before gastrectomy performed; Days in brackets in Section B refer to days in hospital before onset of hæmatemesis.

to expect the hypertensive to bleed to death more rapidly than the normal person—even this does not correspond with the observed facts as set out in *Table II*. Clinically and pathologically we are continually brought to realize that the governing factor is the speed of advance of the ulcer process; it is impossible to believe that at present we know what controls this.

Why are the gastric arteries almost immune from the ordinary forms of arteriosclerosis? In aortic stenosis the aorta is presumably protected from stress by the stenosed valve; seen at autopsy it is strikingly free from atheromatous lesions. Is it possible that the gastric arteries are protected from stress by their anastomoses? Bentley and Barlow (1952) have shown that there are no less than five series of anastomoses between the vessels: (1) between the main trunks of the right and left gastric and gastro-epiploic arteries; (2) a submucous plexus formed from anastomoses between the main penetrating arteries; (3) an arteriovenous submucous anastomosis; (4) anastomoses between the subsidiary branches of the main submucous plexus before the origin of the mucosal arteries; and (5) anastomoses of the vessels to the mucosa itself. The second, third, and fourth series are absent in the immediate region of the lesser curvature.

THE ARTERIAL LESIONS NEAR PEPTIC ULCERS

Arterial lesions are common and well known in the region of peptic ulcers. Because they are not the ordinary forms of arteriosclerosis they will be called 'chronic erosion', 'subacute erosion', and 'acute erosion'. Some arteries show combinations of these, especially subacute and chronic.

Some degree of chronic erosion is seen in the arteries near most peptic ulcers removed surgically (*Fig.* 706). But for this most patients with a gastric or duodenal ulcer would bleed to death. The arterial lumen in chronic erosion is more or less occluded by mucinous infiltration of the intima. The mucus is the mesenchymal type with a high molecular ester sulphuric acid, it gives metachromatic staining with toluidine blue (Cameron, 1952). The elastic laminæ are not affected. Calcification is the greatest rarity. There is no cell exudate and no periarteritis. It is quite common to see the lumen reduced in size for upwards of 0·5 cm., and sometimes it extends for 1·0 cm. or even farther. Chronic erosion is best developed in ulcers which are genuinely chronic—as distinct from recurrent. The impression that chronic erosion is better developed in gastric than duodenal ulcers is probably wrong and depends on the larger number of gastric ulcers available for satisfactory study in surgical material. It is a distinctive lesion which has a limited distribution in disease of other organs: it is often well developed in the arteries of a gall-bladder with a subsiding acute inflammation; in the arteries of the uterus—especially the cervix; in the arteries leading to a tuberculous testis; sometimes in the arteries passing through a carcinoma of the colon; its occurrence in carcinoma of the stomach will be referred to later. It is possibly the result of the action of some chemical substance which is not directly related to bacteria. It is not seen in the arteries of an acutely inflamed appendix nor in other frankly purulent inflammations.

In subacute erosion the lumen of the artery close to the opened part is filled with an organizing thrombus; except for this the artery is of normal structure. The thrombus is variable in length but often less than 1 mm. Occlusion of the arterial

FIG. 706.—Extensive chronic arterial erosion in duodenal ulcer of a man, aged 36. Perforation, 1940; partial gastrectomy, 1950. Note normal internal elastic lamina and media. The degree of reduction of lumen size must only be estimated in transverse sections—here seen in two places to be about one-twentieth of the normal. (× 2.)

lumen is the result of varying amounts of two processes: (1) a fibrin and platelet clot derived from the circulating blood—this is often quite small or even absent; and (2) the development of a syncytial mass of reticulo-endothelial cells from the intima—these are often separated by a surprising amount of mucin. Mucin must be a key substance in the natural mechanism for stopping hæmorrhage from peptic ulcers. Good evidence that the ulcer process has ceased to advance is given by finding many of these subacutely eroded ends covered with recently formed granulation tissue. Surgeons who have discarded the old belief in the danger of 'blowing the clot' from such vessels by transfusion are almost certainly correct—pathologically as well as clinically (*Figs.* 707, 708). Recurrent hæmorrhage depends on digestion of the end by recurrence of the rapid ulcer process, or on the opening of a different artery.

Acute erosion is the typical lesion of fatal hæmorrhage from peptic ulcer. It is naturally rare in routine surgical material but has been found (*Fig.* 709). The ulcer process is almost certainly a rapid one. In sections the ulcer is seen advancing in a relentless line, destroying everything as it goes (*Figs.* 712, 714, 718, 720). Because there is no time for the development of chronic erosion the arteries opened are normal in structure. It is at least close to the truth to state that only a normal artery can give rise to fatal hæmorrhage. Acutely eroded

arteries often stand up in the floor of the ulcer; this is the traditional and only reason for (1) calling them 'arteriosclerotic', and (2) stating that hæmorrhage continues because the 'sclerosed artery' cannot contract. The artery stands up for two reasons: (1) the elastic laminæ are somewhat more resistant, or rigid when necrosed (*Fig.* 718); and (2) the artery is frequently opened on one side—this is important because both ends will spurt blood on account of the free anastomoses (*Figs.* 715 and 722). The acutely eroded artery is sometimes opened along a considerable length.

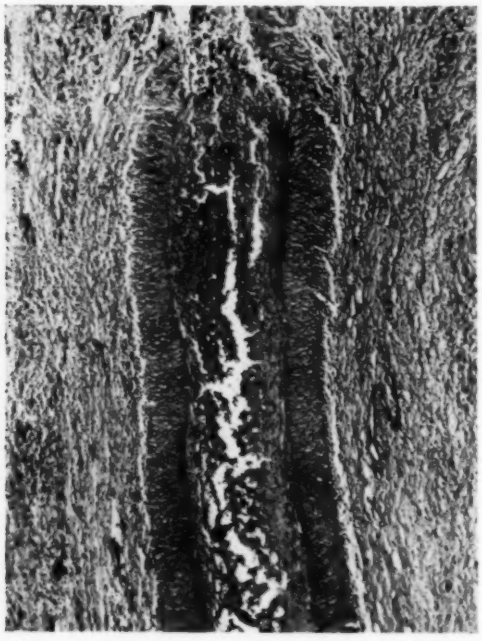

FIG. 707.—Subacute arterial erosion in gastric ulcer of a woman, aged 41. Partial gastrectomy. Internal elastic lamina and media normal. Lumen mostly obliterated by rapidly proliferating reticulo-endothelial cells and mucinous stroma. Note impossibility of 'blowing the clot' by transfusion. (× 11.)

FIG. 708.—Subacute arterial erosion in the chronic perforating gastric ulcer of a man aged 51; gastrectomy specimen. Media and internal elastic lamina normal. Lumen partly closed by small thrombus, mostly by proliferating reticulo-endothelial cells in abundant mucinous stroma. Note virtual impossibility of 'blowing the clot' by transfusion. (× 11.)

FIG. 709.—Acute arterial erosion in gastric ulcer of a man aged 54. In hospital thirteen days before partial gastrectomy; admitted for pain. At least two-thirds of the artery is necrotic. Gastrectomy obviously performed a very short time before onset of massive hæmorrhage. Note absence of reaction in artery and impossibility of its contraction to lessen bleeding. Cases such as this are ascribed to the 'stress reaction' of entering hospital; they prove our lack of fundamental knowledge about the ulcer process. (× 11.)

Two main types of ulcer are associated with massive hæmorrhage. The first is shown in *Figs.* 715–717. These are flat, shallow, and often large; typically there is no indication of their presence from the peritoneal surface; they are gastric rather than duodenal. The second type is duodenal rather than gastric, but is not infrequently prepyloric as in *Fig.* 710. This is an ulcer of chronic perforating type with an acutely eroded artery (usually duodenal) in its base—there has presumably been a sudden speeding up in the ulcer process. Why some ulcers advance rapidly and others slowly is not known; a hint is given by Stewart, Hale, and Hix (1949) who found that "in the group of patients with malnutrition and avitaminosis, as in those with chronic alcoholism, the peptic ulcers were found to be especially large". The obvious seriousness of hæmorrhage which recurs after a few days may have a similar explanation—though the picture is then even more apt to be complicated by uræmia, etc.

Because I have never seen a case of 'gastrostaxis' I do not believe it exists. Cases such as those shown in *Figs.* 715–717 could easily have been diagnosed as 'gastrostaxis'. In massive hæmorrhage the mucosa is covered generally with tenacious mucus which is blood-stained to an appreciable degree; this obscures the shallow ulcer, which may

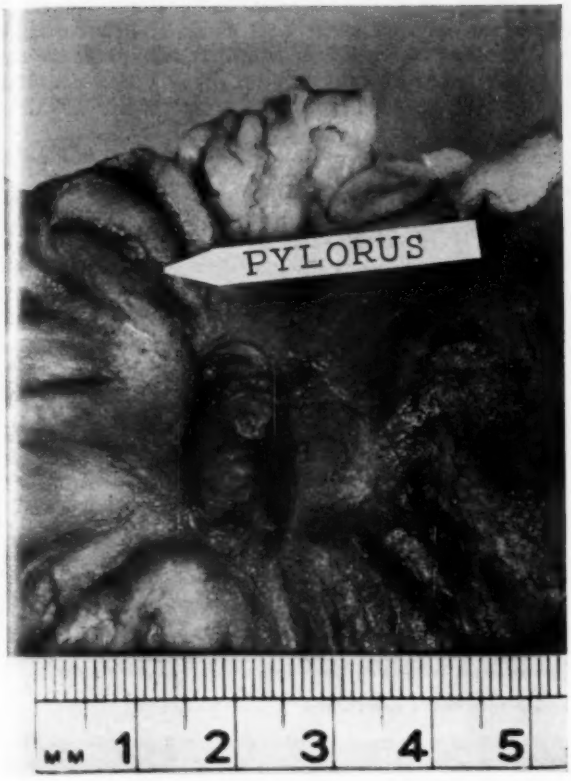

FIG. 710.—Prepyloric ulcer from woman, aged 54, dying in seventeen hours of massive hæmorrhage from acute erosion of artery in base. Blood-urea at eleven hours, 47 mg. per cent; at seventeen hours, 114 mg. per cent. Heart weight, 20 oz. Out of a total of 26 cases she was 1 of the only 8 cases with evidence of hypertension.

FIG. 711.—Ulcer with acutely eroded artery shown in *Fig.* 710. Serial section No. 51. The acute erosion is shown in sections No. 23 143. (× 2.)

FIG. 712.—Enlargement of artery shown in *Figs.* 710, 711. All except the lowest part is necrotic. Note complete absence of arteriosclerosis and impossibility of contraction to control hæmorrhage. There is no clot to 'blow' by transfusion. (× 11.)

FIG. 713.—Man, aged 58. Massive gastric hæmorrhage three days ago; recurrence previous day. Died on table before partial gastrectomy performed. Acute erosion of previously normal artery. All except lowest part of artery is necrotic. Weight of heart, 26 oz., aortic regurgitation. (× 11.)

be exceedingly difficult to find. I would sooner believe that I had failed to discover the artery of fatal hæmorrhage than that 'gastrostaxis' exists.

Among the 26 fatal cases recorded in *Table II*, 2 were the result of massive hæmorrhage from duodenum so that the ulcerated surface no longer faced into the lumen but could be covered by peritoneum outside of the lumen. Ulcers so covered have rarely if ever continued to bleed, whereas ulcers left in the lumen where they are covered by duodenal

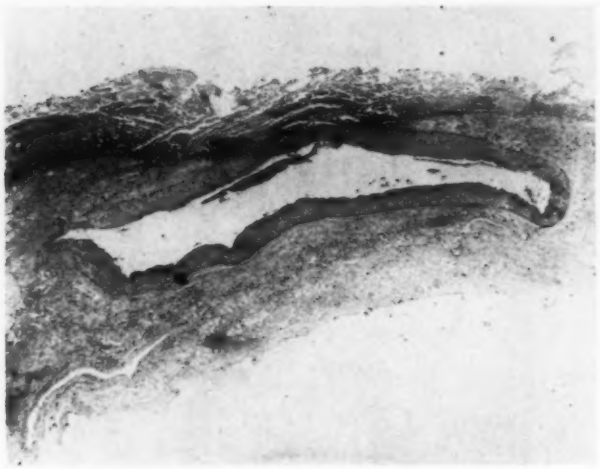

FIG. 714.—Nearby part of artery shown in *Fig.* 713. Showing how progression of the acute erosion opens more of a previously normal artery and so prevents cessation of hæmorrhage. Contraction of artery impossible. (× 11.)

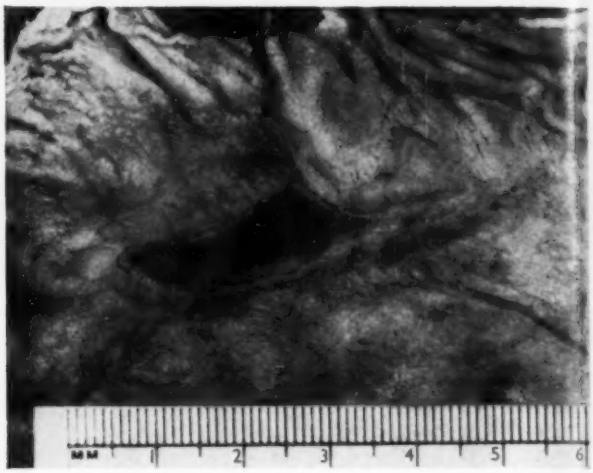

FIG. 715.—Gastric ulcer from a man, aged 89. Acute arterial erosion caused death in one day; patient had been in hospital for fifteen days before this. Shallow ulcer not seen from peritoneal surface. Because of free anastomoses both ends would spurt blood.

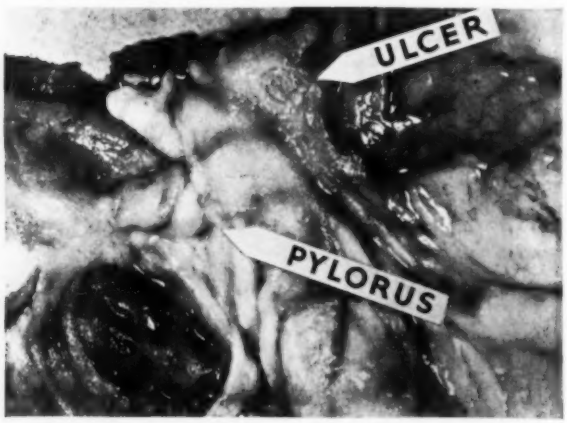

FIG. 716.—Shallow prepyloric ulcer not recognizable from the peritoneal surface in a man aged 49. Fatal hæmorrhage from acute arterial erosion in 30½ hours. Heart normal.

FIG. 717.—Shallow ulcer near œsophagus not recognizable from the peritoneal surface in a woman aged 77. Subacute erosions of multiple arteries of submucous plexus gave frequently recurring hæmorrhages. Uræmic death. Heart normal. Ulcers such as those shown in *Figs.* 715–717 are difficult to find even post mortem. The mucosa generally is covered with bloody mucus; this obscures the real lesion and gives rise to the erroneous concept of 'gastrostaxis'.

duodenal ulcers two days after gastrectomy. This is a well-known occurrence when the ulcer itself has not been removed. To prevent this complication Porter, Harvey, and Schullinger (1950) 'exteriorize' the ulcer: "Because of the danger of secondary hæmorrhage from the ulcer if it is left in the gastrointestinal tract, we consider it more important than we once did to exteriorize it, transfixing the bleeding point if one is found." Harvey (1950) states: "The common dangers following emergency resection for acute hæmorrhage do not include death from operative shock if the case has been well managed, but stem from difficulties in properly treating the duodenal stump, secondary hæmorrhage from the ulcer if it is not exteriorized from the lumen, and thrombophlebitis with its sequelæ". Harvey (1952) also writes, "By exteriorizing the duodenal ulcer, we meant removing the ulcer bed from the wall of the contents have often bled again. We have lost several patients from a secondary hæmorrhage of this kind."

THE GASTRIC ARTERIES IN CARCINOMA OF THE STOMACH

It was thought desirable to examine the truth of the common teaching that chronic erosion is a lesion of arteries in ulcer but not in cancer of the stomach. Cases of ulcer-cancer, where there was satisfactory evidence of pre-existing peptic ulcer, were separated because they might be expected to have such lesions. The cancers were divided into three groups: (1) Diffuse infiltrating—this has little

o no mucosal ulceration, is the type which often gives negative occult blood-tests in the fæces, and in some cases progresses to ' leather-bottle stomach ' ; these should be the least likely to show arterial

FIG. 718.—Male, aged 59, in hospital four days before the onset of hæmorrhage which proved fatal in two days. Acute erosion of duodenal artery. Note darkly stained normal internal elastic lamina and normal media. (× 11.)

lesions ; (2) Adenocarcinoma, often massive growths with much sepsis but little ulceration ; (3) Ulcerated cancer—these growths have many of the gross features of a peptic ulcer, but have tumour tissue

Table III.—GASTRIC ARTERIES IN CARCINOMA OF STOMACH

A. In Ulcer-cancer (8 *Cases*):—
3 females, aged 37, 61, 70 (average 56 years)
5 males, aged 62, 63, 65, 68, 70 (average 66 years)
Arteries normal, 2 : F. 37 ; M. 70
Chronic erosion, 5 : F. 61 ; M. 62 ; M. 63 ; M. 65 ; F. 70
Subacute on chronic erosion, 1 : M. 68

B. In Carcinoma (20 *Consecutive Cases*):—
10 males, aged 36–67 (average 55 years)
10 females, aged 32–77 (average 55 years)
1. Diffuse infiltrating carcinoma (8 cases) :—
Normal arteries, 7
Chronic erosion, 1
2. Adenocarcinoma (8 cases) :—
Normal arteries, 4
Chronic erosion, 4
3. Ulcerated carcinoma (4 cases) :—
Normal arteries, 2
Subacute erosion, 1
Chronic erosion, 1

throughout the floor. The results, set out in *Table III*, show that chronic erosion is anything but rare in carcinoma of the stomach.

DYNAMICS OF HÆMORRHAGIC SHOCK. DEATHS FROM URÆMIA

Continuous or recurrent hæmorrhage is by no means the only cause of death in patients with massive hæmorrhage from peptic ulcer. Of the 26 fatal cases 6 were uræmic and others almost so. The gross appearances in the lungs were mainly those of œdema ; microscopically this was complicated by bronchopneumonia but as this is thought to be secondary in most cases emphasis is accordingly placed on the uræmia. Uræmia directs attention to fundamental pathological processes, especially tissue anoxia from blood-loss and shock. This is emphasized by Porter, and others (1950) in their excellent diagrammatic representation of the mechanics of hæmorrhagic shock. In brief : " tissue anoxia is the root of all evil " :—

FIG. 719.—Male, aged 77. Subacute erosion of duodenal artery which bled twenty-one days before onset of fatal hæmorrhage. (× 11.)

FIG. 720.—Male, aged 77, recurrent hæmorrhage after twenty-one days. The acutely eroded duodenal artery ' stands up ' in the floor of the ulcer ; it is not arteriosclerotic. (× 2.)

a. In most cases hæmorrhage is associated with rapid compensatory mechanisms and there is prompt recovery.

b. In very few cases hæmorrhage is so rapid and shock so deep that death occurs before treatment is possible, e.g., *Case* 1, Section B, in *Table II*.

c. Decompensated shock may develop as a result of continuing hæmorrhage plus various factors which reinforce shock, e.g., fear, dehydration, malnutrition, previous anæmia, etc. Tissue anoxia then affects

many organs, causing a variety of clinical and chemical effects. The chemical effects are often spoken of as the 'electrolyte balance', the correction of this sometimes takes far too prominent a part in management. The most important observation is the blood-urea, and after this the blood-chlorides. Adding

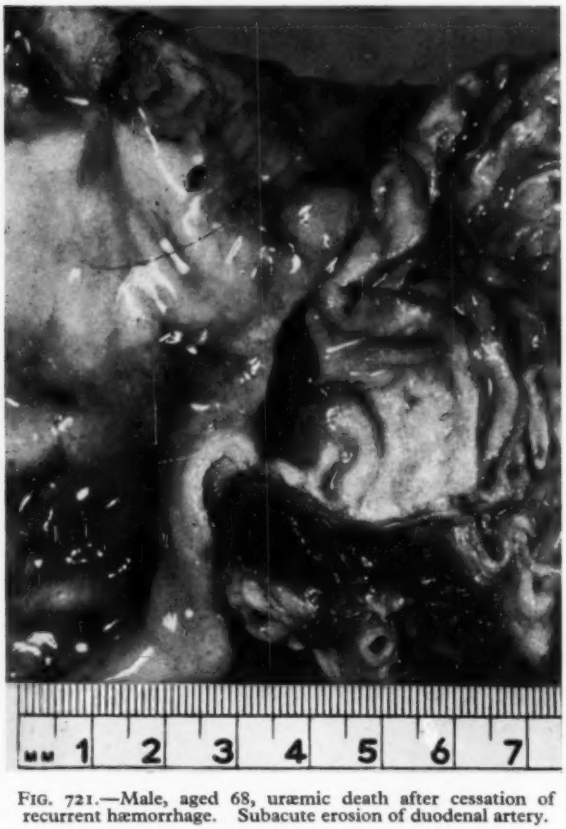

FIG. 721.—Male, aged 68, uræmic death after cessation of recurrent hæmorrhage. Subacute erosion of duodenal artery.

sodium or potassium to the blood is easy enough, but it is a pitifully small part of the problem facing us. In the stage of decompensated shock three courses are open: (1) with prompt and adequate treatment recovery is usual but the patient is of 'low resistance type'; (2) with delayed or inadequate treatment shock may continue for days and recovery is doubtful; (3) with no treatment, or good treatment in a very poor risk patient, irreversible shock is likely to lead to death.

The frequency of the uræmic state from 'shock nephrosis' after massive hæmorrhage is an important reason for adequate treatment from the onset. There is no reason to make any real distinction between these patients and those with 'traumatic uræmia' after road accidents, or the less recognized uræmic state which so often follows a ruptured ectopic gestation.

THE HISTORY IN MASSIVE HÆMORRHAGE FROM PEPTIC ULCER

One of the most striking features of the history of fatal cases is the absence of pain and this suggests the possibility that there is a relationship between pain and chronic arterial erosion.

1. Male, aged 49; ulcer shown in *Fig.* 716. Heart normal. "Eight weeks ago had 'black vomit', but no pain. 8.30 p.m. yesterday: 'black vomit', about 1 pint, dizzy, stools very dark. No pain. 'Never had stomach trouble'. 2nd day (hospital): B.P. 110/60, R.B.C. 2·9 million, blood-urea 65 mg. per cent. Transfused 4 pints Group O Rh +ve. 3rd day: died 3.5 a.m., 30½ hours after onset."

Absence of pain is not so common in duodenal as in gastric hæmorrhages, the suggested reason is that here the typical pathological process is the rapid development of acute erosion in a chronic perforating ulcer, the pain is probably correlated with the latter.

2. Male, aged 59, acutely eroded duodenal artery shown in *Fig.* 718. Heart normal. "He was admitted with severe epigastric pain. He seemed to be responding to dietetic treatment quite well, but after being in hospital 'several' (four) days he started with melæna followed by severe hæmatemesis. Despite transfusion of 10 pints of blood, the bleeding never ceased and operation was not feasible. He died from the hæmorrhage after two days."

Development of such a massive hæmorrhage after four days on 'modern medical treatment' in hospital should convince us that we really do not

FIG. 722.—Male, aged 66, uræmic death after cessation of hæmorrhage. Subacute erosion of duodenal artery. This artery had been normal where eroded, 2 cm. away near the pancreas it had a small calcified atheromatous plaque—this had not prevented the hæmorrhage from stopping. (× 11.)

know what governs the healing or progression of an ulcer. Another example with an even more rapid hæmorrhage is:—

3. Male, aged 57. Heart normal. "This patient was admitted on account of pyloric stenosis due to his duodenal ulcer. Two days after admission, in spite of therapy to improve him, he had a very severe hæmorrhage during the night and died very rapidly."

Patients with massive hæmorrhage from peptic ulcer, like those with an intestinal obstruction, are nearly always worse than they appear to be clinically,

death often comes as a surprise to the attendant and is commonly described as "sudden". The reason for this is that the death is so largely chemical and "extrarenal uræmia" hardly betrays its existence clinically. In the two histories which follow the first had a normal blood urea on admission, later observations were not made, the second had no estimation at any time unfortunately.

4. Male, aged 34. Heart hypertrophied, 15 oz. "With regard to this man, he improved from his hæmatemesis for a while, and we debated whether we ought to advise operation. A surgical colleague saw him with me, but we thought the best thing was to leave him and hope for a better improvement. Unfortunately he died quite suddenly (after four days). The post-mortem examination showed a chronic duodenal ulcer, which was surprising with the very short history he gave of pain."

5. Female, 60 years. Heart normal. "This patient, who was admitted for hæmatemesis (not pain), continued to bleed and in spite of repeated blood transfusions she died two days after admission. The post-mortem examination showed a small acute ulcer less than 1 cm. in diameter high up on the lesser curvature with the bleeding artery in its centre. Looking back there was some improvement in her condition following the first two pints of blood, but then she relapsed badly on the second day, and by then the surgeon decided she would have died on the table if surgical methods had been attempted. These cases are very alarming and disappointing."

The uræmic state may develop rapidly, as in the case illustrated in *Figs.* 710–712, or fairly slowly as in the following case which was painless throughout:

6. Female, 77 years. Heart normal. "Vomited a lot of blood this a.m., also melæna. Very pale. B.P. 70/50. R.B.C. 4 million. Group A, Rh +ve. Transfusion 3 pints blood. 3rd day: Fresh hæmatemesis. Shocked. Blood transfusion 1 pint. 6th day: R.B.C. 3·9 million, blood-urea 90 mg. per cent. 8th day: Hæmatemesis again, half pint. B.P. 110/60. 10th day: Keeps on bleeding, likes beer, so we give it to her. She is not going to live for long. R.B.C. 5 million. Blood-urea 244 mg. per cent. 15th day: Blood-urea 300 mg. per cent. Suddenly collapsed, died later." Her ulcer is shown in *Fig.* 717. Pathologically it appears probable that her first hæmorrhage was by far the largest, but ceased; the recurrences were fresh subacute erosions of arteries of the submucous plexus. It is interesting to observe that had she been subjected to laparotomy in the early stages the surgeon would have seen no lesion and would have done a subtotal gastrectomy; he would have left the bleeding ulcer behind, but the bleeding would probably have ceased. Fortunately such high ulcers are uncommon; some have been known to continue furious bleeding after 'blind' subtotal gastrectomy until death ensued in spite of enormous transfusions. Although this case may be doubtful the series as a whole lends strong support to the view that when the surgeon finds no lesion at laparotomy for hæmorrhage from the stomach or duodenum he should proceed to do a subtotal gastrectomy.

PATHOLOGICAL INVESTIGATIONS AND PRINCIPLES GOVERNING TREATMENT

If chronic erosion is well developed in the arteries of the ulcer the case is suitable for medical treatment because it cannot bleed to death; if acute arterial erosion is present the patient will almost certainly die unless a partial gastrectomy is performed in the pre-uræmic state (i.e., usually within 24, or at most, 48 hours); an intermediate group with subacute arterial erosion may recover under medical treatment or die within a week or two of uræmia or recurrent hæmorrhage. Unfortunately this statement has little practical value because we cannot see the arteries before operation or autopsy. When the factors which govern the progression or healing of an ulcer are known we will be able to expect all cases with chronic and subacute arterial erosion to respond to medical treatment; even then acute arterial erosion will almost certainly be a purely surgical lesion.

There can be no doubt that if all the patients who should have a partial gastrectomy in the pre-uræmic stage are to be operated upon, many who would have recovered without operation will be selected also. With more accurate knowledge the number of these unnecessary and possibly undesirable operations will fall.

Pathological investigations are essential for correct management, but the pathologist must not pretend to knowledge he does not have and must be very careful that his results do not mislead.

The complex dynamics of arterial hæmorrhage combined with shock, and probably associated with other variable conditions such as fear, dehydration, malnutrition, etc., greatly detract from the value of pathological investigations. The figures obtained from blood-counts are nearly always misleadingly high; they lag at least 24 hours behind the true condition. The concept of 'blood-volume' for anything other than a stabilized animal is so naïve that the trouble taken to estimate it can hardly be worth while. The figure obtained after injecting T 1824 may or may not represent the 'blood volume' at the time of the investigation; by the time the result is obtained it is almost certainly meaningless for the state then in existence. The volume of the capillary bed is so great and so variable that we cannot interpret such tests. We are asking for the volume of a concertina. By observing how the capillaries bleed when investigating accident cases it is generally possible to make a reasonable guess at how much should be subtracted from the observed red-cell count because of the shock; this gives a crude but clinically satisfactory guide to the need for blood transfusion. The 'reasonable guess' may require modification in the light of follow-up blood examinations.

The rule of Stewart, Hale, and Hix (1949) to operate if the "total circulating red-cell mass was reduced to 60 per cent, or less, of normal" is a useful guide—as will be seen from a study of *Table II*. There are two serious objections to this rule: (1) in the type of case under consideration I do not believe we can estimate the figure with any degree of accuracy; and (2) it does not take into account the time taken to lose 40 per cent or more of the red-cell mass.

There is no doubt that it is very difficult to answer the question "Has the bleeding ceased?". Pathologists asked this question within the first day or so will probably agree with Stewart, Massover, Potter, and Schaer (1948) "that it is impossible to tell which patient will survive on a non-surgical regimen and which will die." Because so many do recover without emergency gastrectomy most will

try to agree with Harvey (1950) that " It appears that the plan of selecting patients for operation among the massive bleeders is feasible."

Patients with massive hæmorrhage may be a poor operative risk for two reason: (1) Because so many are middle-aged or old, about one-third can be expected to have associated disorders. This has usually been given as a reason for withholding surgery, but the attitude of Porter, Harvey, and Schullinger (1950) is better: " Within limits of reason, we recommended operation soonest in patients who were poor operative risks or presented difficult technical problems. An error in judgement in not seizing a fairly good opportunity to operate on them might be irreparable." (2) Because arterial hæmorrhage has continued too long—the mistake most to be feared.

Consideration of all points leads to the following conclusions for management :—

1. Hæmorrhagic shock must be corrected rapidly. If not death may occur from: (*a*) Straight hæmorrhage; (*b*) Delayed shock nephrosis.

2. Selection of those with over-rapid hæmorrhage for immediate operation :—

a. Absence of pain before hæmorrhage;

b. Rapid fall in R.B.C. (figure corrected clinically);

c. Early rise in blood-urea;

d. No obvious response to 1–2 pints of blood.

3. Selection of those with associated disorders for early operation.

4. Selection of those with recurrent hæmorrhage for early operation.

5. Preparation of all others for operation :—

a. Most respond so well that operation not performed, determine R.B.C. and blood-urea at least daily;

b. Response unconvincing, operate within 24–48 hours.

There is, of course, one important proviso: the team available must be adequate. Harvey (1952) writes: " Our failures in most cases have been due to going in too late. This in turn was usually due to lack of teamwork between physician and surgeon. We regard this teamwork as the foundation of the whole system of selecting patients for operation. A good team has good results; an inexperienced team does not do so well."

This paper does not attempt to consider the differential diagnosis of massive hæmorrhage from œsophageal varices, etc.

SUMMARY

1. The gastric arteries at all ages are very nearly immune from the usual forms of arteriosclerosis. Arteriosclerosis cannot play any part in massive hæmorrhage from peptic ulcer.

2. In peptic ulceration the arteries may suffer chronic, subacute, or acute erosion. Chronic erosion prevents massive hæmorrhage. Acute erosion is the typical lesion of fatal hæmorrhage.

3. The type of arterial erosion present and the course of the hæmorrhage are determined by unknown factors which govern the rate of the ulcer process.

4. Chronic arterial erosion is quite common in carcinoma of the stomach; its frequency varies in different types of carcinoma.

5. Hæmorrhagic shock must be controlled rapidly for various reasons. The old belief in the danger of ' blowing off the clot ' by transfusion is based on false pathological concepts.

6. Acute erosion is almost certain to be fatal unless the patient is treated by subtotal gastrectomy, after adequate preparation, in the pre-uræmic stage. There can be no ' medical treatment ' for acute erosion.

7. The probable existence of acute erosion can be diagnosed in various ways, but especially by the history and rate of blood-loss.

8. Many gastric ulcers with acute arterial erosion cannot be recognized at operation. This justifies ' blind ' subtotal gastrectomy.

9. Duodenal ulcers which cannot be removed should be ' exteriorized ' to prevent recurrence of the hæmorrhage.

10. Pathological investigations are essential but require judgement in interpretation if they are not to mislead.

11. ' List cases ' may develop acute erosion after admission to hospital; this is ascribed to the ' stress reaction ', but requires investigation.

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THE INCIDENCE OF ULCER-CANCER

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THE frequency with which carcinoma arises in a chronic gastric ulcer has been a matter of debate for many years. It is not our purpose to review the literature, apart from observing that the incidence has been variously estimated as high as 69 per cent (McCarty and Broders, 1914) and as low as 3·75 per cent (Newcomb, 1932). Since Ewing (1918) showed that the high figures of the Mayo Clinic were due to misinterpretation of heterotopic clumps of epithelium in the margins of healing ulcers (29 per cent in Newcomb's series) it has been generally accepted that the lower figure is a closer approximation.

It might be thought that after the excellent papers by Ewing (1918), Dible (1925), Stewart (1931), and Newcomb (1932) little remained to be added on the subject of the incidence of ulcer–cancer. However, the subject has recently been re-introduced by no less an authority than Sir Heneage Ogilvie (1952), who asserts that despite the pathologist's reports, he is unable to convince himself that malignancy ever arises in a previously benign gastric ulcer. He specifically rejects 'spatial propinquity' as a proof of causal relationship, adding that if there is a relation, it is the cancer that causes the ulcer, and not vice versa; and observes that in cases diagnosed pathologically as ulcer–cancers the age of onset of symptoms has been unusually late, and the symptoms themselves not typical of a benign ulcer.

There is, of course, no theoretical reason why carcinoma should not arise in a chronic gastric ulcer, and it is our belief that this sometimes occurs, although the frequency of the occurrence is difficult to assess accurately. We have seen only 2 cases in the course of seven years at Westminster Hospital, as compared with 218 benign chronic gastric ulcers, and further discussion may be conveniently postponed until these have been presented.

ANALYSIS OF GASTRECTOMY SPECIMENS

In the years March, 1946 to March, 1953, 673 gastrectomy specimens were submitted to the Morbid Anatomy Department by the surgical staff at Westminster Hospital, with the presumptive diagnosis of peptic ulcer or carcinoma. This figure excludes twelve gastrectomies for anastomotic ulcers, pyloric stenosis without visible ulcer, and sundry tumours like myoma or sarcoma. Of the specimens under review 104 showed gastric carcinoma and the remaining 569 could be assigned to one of four categories :—

1. 237 showed a definite gastric ulcer—chronic, subacute, or acute.

2. 96 had a definite ulcer in the duodenal part of the specimen.

3. In 157, no ulcer was found macroscopically and the specimen was discarded without histological examination.

4. In 79, pieces from areas suspicious of ulceration were examined microscopically, but found to be healthy.

In the overwhelming majority of groups (3) and (4) it was known that a duodenal ulcer had either been crushed by clamps, or intentionally left behind; in a few cases, however, it would appear that the diagnosis of gastric or duodenal ulcer, made on clinical or radiological grounds, was not sustained.

The 237 benign gastric ulcers could be further subdivided into :—

a. Acute 4;
b. Subacute 15;
c. Chronic 218.

Of the 104 carcinomas, only 2 could be unhesitatingly diagnosed as ulcer–cancer, although a further 2 were strongly suggestive. That is to say, during a period of seven years there were 218 proved chronic benign gastric ulcers and 2 ulcer–cancers—an incidence of less than 1 per cent; alternatively, of 104 gastric carcinomas, only 2 per cent could be shown to have arisen in a pre-existing ulcer. These figures are summarized in the accompanying table.

ANALYSIS OF 673 GASTRECTOMY SPECIMENS

Proved gastric ulcer		237
Acute	4	
Subacute	15	
Chronic	218	
Proved duodenal ulcer		96
No ulcer seen—no histology		157
No ulcer found on microscopical examination		79
Carcinoma of stomach		104
Ulcer-cancer	2	
? Ulcer-cancer	2	
Other types	100	
TOTAL		673

CASE REPORTS

Case 1.—Male, aged 72 (retired clerk), who had a hæmatemesis in 1933 followed by eighteen years of indigestion. After some weeks in hospital in 1949 for coronary thrombosis, he was given an ulcer régime, which relieved him. Next year, in the course of a routine follow-up, a barium-meal showed a large ulcer high on the lesser curve of the stomach posteriorly, with considerable scarring around it, causing enough distortion to arouse the suspicion of malignancy. X-ray examination was repeated three months later, but though the ulcer was smaller, the suspicion of malignancy remained. The pain was now felt half an hour after meals, sometimes severe enough to double him up, often felt on waking and worst of all in the evenings. A test-meal revealed achlorhydria.

A few weeks later (June, 1951) he was re-admitted for gastrectomy and two-thirds of the stomach was removed. On opening the specimen a typical chronic ulcer was found on the posterior aspect of the lesser curve, 7 × 10 mm. in its diameters.

HISTOLOGY (*Fig.* 723).—A chronic ulcer, quite typical save for the presence of a focus of poorly-differentiated adenocarcinoma 3 mm. in diameter situated in one margin between the muscularis mucosæ and the muscle, which is invaded. The ulcer floor is completely fibrous and contains neither muscle-fibres nor tumour cells. The other margin is that of a typical chronic peptic ulcer.

SUBSEQUENT HISTORY.—He remained symptom-free until June, 1953, when he developed epigastric pain and tenderness. Exploratory laparotomy revealed recurrent growth on the gastric side of the stoma, and metastases

in the liver. Further resection was not attempted and he died four weeks later.

AT AUTOPSY.—There was a tumour at the site of the anastomosis, 5 × 2 cm., from which protruded threads

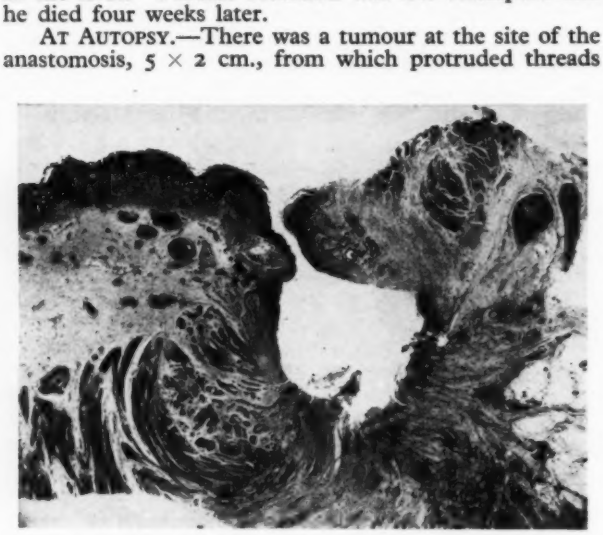

FIG. 723.—*Case* 1. Low-power view of ulcer showing typical fibrous floor, and focus of carcinoma in one wall, between muscularis mucosæ and muscle. H. and E. (× 5·4.)

from the original suture (*Fig.* 724). The liver was riddled with metastases. The tumour microscopically was a carcinoma, considerably more anaplastic than the original growth.

Case 2.—Male, aged 67 (clerk), with ten months' history of epigastric pain two to three hours after meals, relieved by food and alkalis. As he began to lose weight,

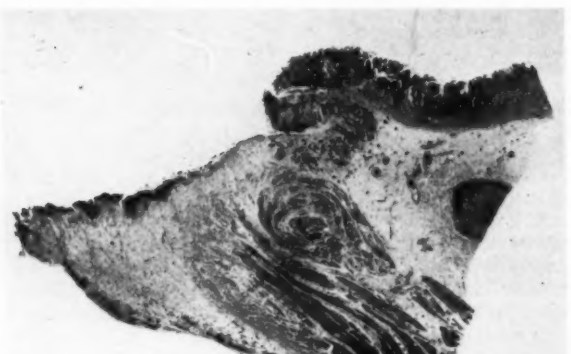

FIG. 725.—*Case* 2. Ulcer-cancer showing focus of carcinoma between muscularis mucosæ and muscle. The wall and floor are typical of a chronic peptic ulcer. H. and E. (× 3.)

a barium meal was performed, which showed active ulceration of the pylorus and an irregularity of the mucosa more suggestive of old ulceration than new growth. A second radiograph six months later revealed a malignant ulcer at the pylorus, not causing obstruction. The test-meal was within normal limits.

A month later total gastrectomy was performed. An ulcer was found in the pyloric antrum, presumed to be malignant, as there were two small metastases in the liver.

HISTOLOGY.—The ulcer (*Fig.* 725) shows malignant infiltration of its wall by a poorly-differentiated adenocarcinoma spreading peripherally between the muscularis mucosæ and the muscle, which is being invaded. The base of the ulcer is densely fibrous, and devoid of muscle-fibres or tumour cells.

SUBSEQUENT HISTORY.—After two months' convalescence he was wasted and had palpable masses in the liver. He died three weeks after going home, and there was no autopsy.

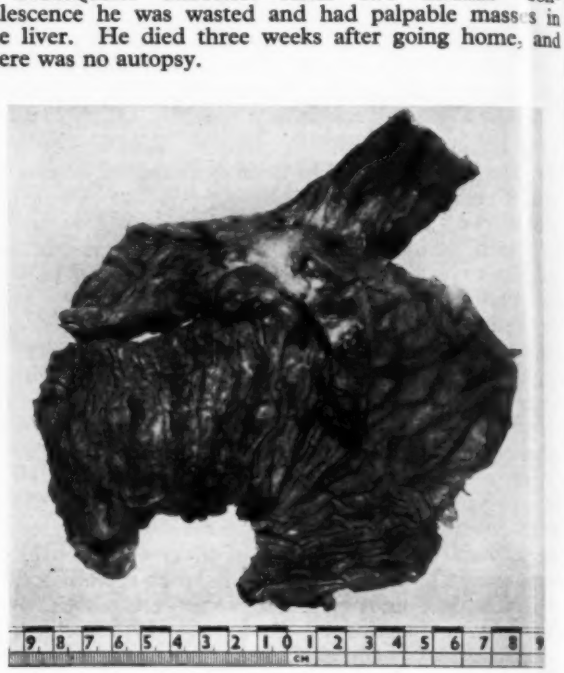

FIG. 724.—*Case* 1. Gastro-enterostomy stoma showing recurrence of growth above the suture line. (× 0·36.)

DISCUSSION

Surgeon and pathologist alike are agreed that ulcer–cancer is essentially a pathological diagnosis, although the clinical and radiological evidence is not to be ignored. Others have mentioned, but have not sufficiently stressed, the fact that it is only in the very early stages of malignant transformation that the diagnosis can be made with confidence. It is essential that the pathologist should be able to demonstrate a malignant focus in the wall of an otherwise typical chronic peptic ulcer; that is, he should be able to disentangle the elements of two separate lesions. If excised too early, the part of the ulcer he examines may not contain the malignant focus; if operation is deferred, the characteristic features of the chronic gastric ulcer may be obscured by tumour. Now if the diagnosis is supportable only in those cases of very early malignancy, it follows that the incidence of ulcer–cancer is higher than our figure of 1 per cent. Just how much higher we do not venture to suggest, nor do we see how an accurate figure can ever be arrived at.

THE PATHOLOGY OF ULCER-CANCER

The microscopical criteria for the diagnosis of ulcer–cancer have been stated often enough, and more recently summarized and well illustrated by Hadfield and Garrod (1947). These are: (1) The floor of the ulcer should be that of a typical chronic peptic ulcer, composed of dense fibrous tissue in which neither muscle-fibres nor tumour cells are found. (2) The muscle should end abruptly near the wall, and between it and the overhanging mucosa should be a focus of unmistakable carcinoma, with evidence of invasion of muscle.

Newcomb stresses the importance of fusion of the muscularis mucosæ with the gastric muscle in

the ulcer wall, believing this to be the only reliable evidence of pre-existing ulceration. Stewart (1931) considers this an exaggerated view-point, and to us it appears that while the sign is valuable when present, its absence does not exclude the diagnosis of ulcer–cancer; in fact, the area of muscular

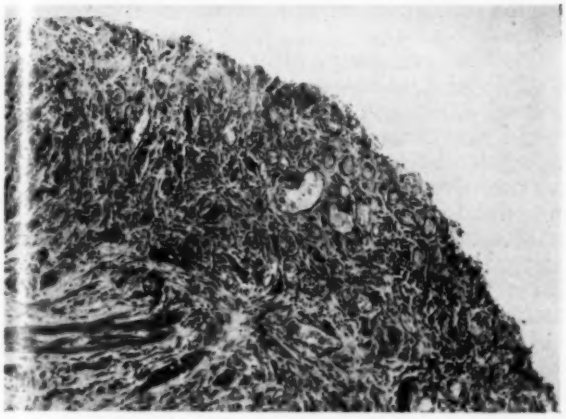

FIG. 726.—Ulcerating carcinoma showing presence of tumour cells and muscle-fibres in the floor. H. and E. (× 45.)

fusion may be destroyed at an early stage by tumour (*see Figs.* 723, 724).

Mention has been made of the danger of mistaking for malignancy the heterotopic foci of gastric mucosa in the margin of a healing ulcer. Text-books of surgical pathology continue to warn against the danger, and it may be as well that they should do so, but the experienced pathologist is not likely to be so deceived.

It is the malignant ulcer with a fibrous base that is misleading. Three of our cases (one adeno-carcinoma, two anaplastic scirrhous) were at first suggestive of ulcer–cancer for this reason, but closer inspection revealed the presence of isolated muscle-fibres or tumour cells in the floor (*Fig.* 726). The presence of muscle-fibres precludes the diagnosis of ulcer–cancer, since these are not found in the floor of a chronic peptic ulcer. The presence of tumour cells alone may invalidate the diagnosis without excluding it as a possibility; for though it may be true, as has been suggested, that tumour cells tend to spread more readily through the surrounding healthy tissue than through the fibrous base of a chronic ulcer, sooner or later the ulcer–cancer is likely to show invasion of its base, and by that time the walls of the ulcer will be destroyed and the diagnosis cannot be more than conjectural. In the two cases still suspected but not proved ulcer–cancer (*see* table) the diagnosis was rejected because of the presence of tumour cells throughout the fibrous base of the ulcer. Both had typical chronic ulcer histories, one of eight years' duration, the other seven.

Ogilvie revives the theory that "all, or the great majority, of these ulcer–cancers are scirrhous cancers which have been attacked by the gastric juice and have undergone the usual process of healing in those parts where all malignant cells have been digested away". There are several objections to this theory. For one thing the malignant focus may be present in one wall of the ulcer and not the other (*Fig.* 723). It is not likely that the digestive juices would destroy the malignant cells so completely on one side of the ulcer, nor that carcinoma would grow in one direction only. It is our experience that in ulcerating scirrhous cancer, tumour cells are found all around the margin

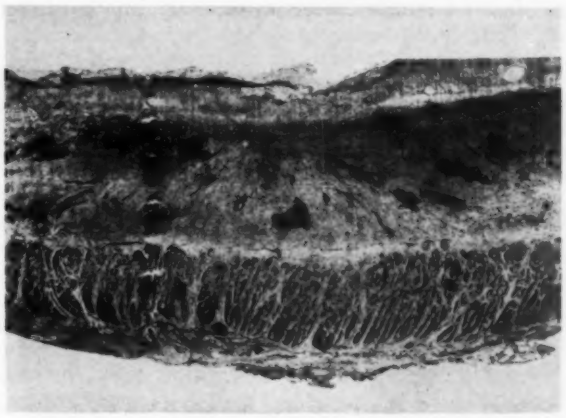

FIG. 727.—Very early scirrhous cancer, not completely ulcerated. The muscle is unbroken, though permeated by tumour cells. H. and E. (× 4·2.)

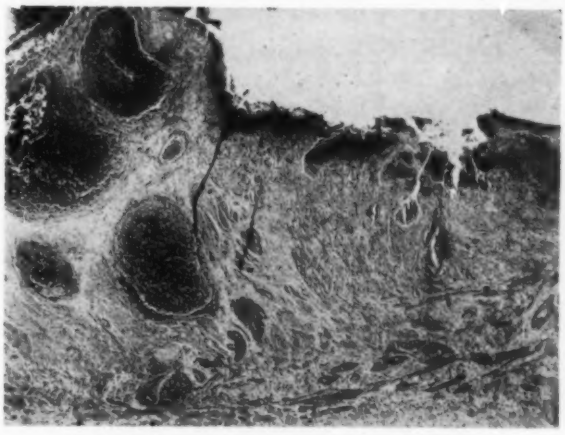

FIG. 728..—Lymphosarcoma associated with gastric ulcer. Note the lymphoid nodules, and the presence of muscle-fibres in the floor of the ulcer. H. and E. (× 3.)

of the fibrous ulcer base, in which, however, remnants of malignancy can always be demonstrated.

A more serious objection is that even in the earliest and most slowly-growing of scirrhous cancers, tumour cells are to be found spreading widely before the overlying mucosa is completely destroyed, and long before the gastric muscle is breached. *Fig.* 727 shows the earliest ulcerating scirrhous cancer of the series. The mucous membrane is barely excavated, the muscle is intact, and yet tumour cells are already in the subserous layer.

The patient was a woman, aged 64, with one year's history of epigastric pain, not definitely related to meals. X-ray examination revealed a lesion of the lesser curve of the stomach suspicious of malignancy, and gastrectomy was performed. She died four years later with abdominal and skeletal metastases.

Dible (1925) describes and illustrates a similar case (No. 22, *Figs.* 419, 420).

This does not mean that carcinomata cannot give rise to a type of peptic ulcer. Mallory (1940) shows clearly that this happens not infrequently; but his photomicrographs are not those of a *chronic* gastric ulcer, but an acute or subacute penetrating ulcer, with muscle extending right to the ulcer margin, and with little fibrosis in the floor. *Fig.* 728 shows a similar ulcer, apparently due to underlying lymphosarcoma.

The patient was a man of 72, who gave a vague history of dyspepsia of five months' duration. At operation, the spleen was found to weigh 690 g., and he died five months after gastrectomy with the terminal blood-picture of lymphatic leukæmia.

The presence of carcinoma and an apparently independent chronic peptic ulcer in different parts of the same stomach is sufficiently rare in our series —2 out of a total of 104 carcinomata—to be regarded as fortuitous. Neither condition is uncommon, and it is not surprising that they should sometimes coexist. In one of these the base of the ulcerating cancer (seen in *Fig.* 726) provided a sharp contrast to the typical collagenous floor of a chronic benign ulcer higher up the lesser curve.

The patient was a man, aged 43, with a two-year history of epigastric pain half an hour to one hour after meals, relieved by alkalis and vomiting, and with typical remissions. Barium meal showed an ulcer crater in the lesser curve and another in the prepyloric region, the latter suspicious of malignancy.

Now, if it is uncommon to find a chronic benign ulcer and a carcinoma in the same stomach, it should be fantastically rare to find cancer arising in the wall of a chronic ulcer unless there is a causal relationship. Yet we know that this happens from time to time. The occurrence of two different diseases in the same organ may be described as 'spatial propinquity', but when the one occurs *within* the other, the pathological evidence for causal relationship is at least as strong as that of, let us say, Marjolin's ulcer.

CLINICAL CONSIDERATIONS

Age Incidence.—In Dible's series the average age of the patient at the time of operation was 44 for benign ulcer, 49 for carcinoma; the age of onset of symptoms being 33 and 45 respectively. In Newcomb's six ulcer–cancers the average age at the time of operation (i.e., early in the evolution of the tumour) was 45, the age of onset of symptoms being 39.

It has been argued that this onset of symptoms at a later age indicates that the ulcer was not an ordinary peptic ulcer, but malignant from the outset. But there is a generous overlap in the ulcer and ulcer-cancer age groups, and it is equally arguable that as age is a factor in most cancers, it is hardly surprising that the small percentage of ulcers that become malignant should occur in the older age groups.

Patient's History.—It is obviously an advantage, in diagnosing ulcer–cancer, that the history should be compatible, but the absence of a typical history should not exclude the diagnosis. The symptoms of peptic ulcer may be fairly characteristic, but are not always elicited in patients who have an undoubted ulcer at autopsy; nor is it rare to find an intact mucosa in some gastrectomy specimens resected for suspected gastric ulcer. The same observations apply, though to a lesser extent, to gastric carcinoma.

However, an atypical ulcer history is not the rule in ulcer–cancer. Stewart, who found 6 per cent of ulcer cancers in 277 chronic peptic ulcers, would not diagnose the condition without clinical confirmation; and in Newcomb's series the history of chronic ulcer was typical enough in all his cases.

SUMMARY

Ulcer–cancer is a definite entity and occurs in at least 1 per cent of chronic gastric ulcers. As it can be diagnosed only in the very early stages of malignancy, its incidence is certainly higher, though an accurate figure cannot be assessed. Two new cases are described.

The criteria of diagnosis are reviewed. The finding of undoubted cancer in one margin of an otherwise typical chronic ulcer is regarded as evidence of a causal connexion. The presence of muscle-fibres or tumour cells in the base of the ulcer invalidates the diagnosis.

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PANCREATICO-BRONCHIAL FISTULA

A REPORT ON TWO CASES

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Pancreatic fistula is a well-known but uncommon surgical condition. The majority of recorded cases have followed the treatment of cysts, but they can also result from injury, or after operations on the stomach, biliary tract, spleen, and left kidney, or when the abdomen has been opened in cases of acute pancreatitis.

Usually the fistula opens on to the abdominal wall by a single sinus via the operation wound or the site of a drainage-tube. It is estimated that 80 per cent of them heal spontaneously and few surgeons have had the need to treat many by operation.

Many excellent contributions to the literature on the treatment of pancreatic fistula have been made in the past, notably by von Mikulicz (1903), Schmeiden and Sebening (1928), Stern (1930), and more recently by Warren (1951), and Aldis (1946); but I have been unable to find any record of a pancreatico-bronchial fistula.

It is, therefore, interesting to record the following 2 cases treated by me in the last year in which the fistulæ discharged into a bronchus as well as on to the abdominal wall.

It may prove useful to consider the points of difference and similarity between them, and discuss the treatment which led to their cure.

The diagrams (*Figs.* 730, 731, 733, 734) were compiled from the study of many radiographs and examinations in the dissecting room. They may help to clarify photographs and prints which do not always lend themselves to reproduction.

CASE REPORTS

Case 1 (*Figs.* 729-732).—On March 25, 1952, a soldier, aged 26, received a crush injury of the abdomen whilst serving in Korea. Laparotomy was performed on the day of injury, when a laceration of the liver was found and sutured. The gall-bladder and ducts were intact, but a large retroperitoneal hæmatoma in the region of the pancreas was noted. The abdomen was closed with a drain in the wound.

On March 31 (six days later) the drain was removed, but an intermittent discharge of clear yellowish fluid which digested the skin continued. During the periods of free discharge he complained of abdominal colic, thirst, and a greatly diminished flow of urine.

On April 13 aluminium paste was applied round and over the sinus. The discharge stopped but was followed by an increase in pain in the left hypochondrium and in the left shoulder. During the next few days the temperature began to swing, the pain became severe, and radiographs showed a raised and fixed diaphragm on the left side.

On April 19 an abscess at the site of the drainage-tube was opened, and 1½ pints of pus were evacuated. This became clear fluid which continued to drain at the rate of 500 to 600 c.c. a day.

On April 23 the left subphrenic spaces were explored through a subcostal incision in front and by resecting the 12th rib at the back. No abscess was found. At this stage his notes record a urinary diastase of 50 units.

He remained ill during May and June, with pain in the left hypochondrium and left shoulder, and he had difficulty in swallowing. Large quantities of pancreatic fluid continued to discharge from the fistula.

On June 30 he arrived at The Queen Alexandra Military Hospital, Millbank, having been evacuated from

Fig. 729.—A sinogram with lipiodol outlines a cavity and some of the pancreatic ducts.

Japan by air. During the flight the fistula became blocked and he was greatly distressed by a cough with profuse frothy sputum and hæmoptysis. When he landed the discharge returned, with some relief of symptoms.

On arrival he was extremely emaciated and the fistula was discharging profusely. Excoriation of the skin extended over the lower abdomen and into the perineum and round the scrotum. A multitude of lymph-glands in the groins, the abdominal wall, the intercostal spaces, and the axillæ had enlarged and were acutely tender. His morale was very low and it was difficult to get his co-operation.

On July 7 *the sputum was found to contain pancreatic ferments.* A sinogram revealed a pocket of concentrated dye just deep to the anterior abdominal wall, and outlined some of the pancreatic ducts (*Fig.* 729), whilst deep in the left hypochondrium a large irregular pool of less concentrated dye was seen.

Each time the abdominal fistula closed, the volume of sputum increased. A diagnosis of pancreatico-bronchial fistula was made and operation advised.

The objects of the operation were: (*a*) to define the fistulæ, and (*b*) to drain the main collection of fluid either by anastomosing the abdominal fistula to some part of the upper abdominal tract, or by a direct dependent drain through the abdominal wall.

On July 15 a laparotomy was performed through a thoraco-abdominal incision which encircled the fistula. It proved a complete failure.

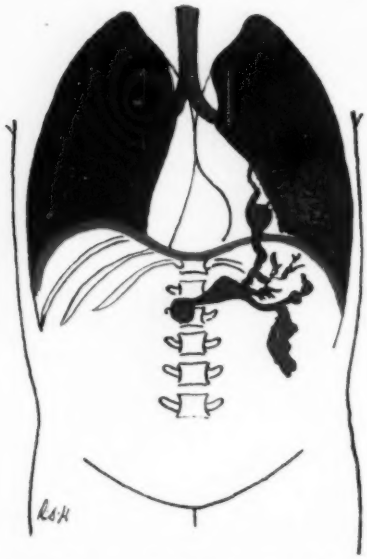

FIG. 730.—*Case* 1. A diagram compiled from radiographs and operation findings. The diaphragm is shown in red. The green areas show the path of spread of escaping pancreatic fluid.

FIG. 732.—Radiograph with lipiodol showing the abdominal fistula with an upward extension through the diaphragm and the production of a bronchogram.

The fistula, which had been injected with Bonney's blue, pierced the lesser omentum and became lost in a dense maze of adhesions. It was friable and quite unsuitable for any anastomosis. The pancreas was narrowed and fibrotic where it lay across the vertebral bodies and the tail end lay in a cavity containing fluid, an extension of which tracked upwards between the stomach and spleen towards the diaphragm. The lower lobe of the left lung was adherent to the diaphragm and was indurated and hard, with an area of consolidation in its centre. It seemed unwise to interfere, so a drain was placed in the most dependent part of the peripancreatic cavity and the abdomen was closed. For some days after the operation the sputum was stained blue.

FIG. 731.—*Case* 1. A lateral view shows the relationship of the escaping pancreatic fluid, shown in green, to the stomach and diaphragm, shown in red.

Continuous suction failed to reduce the volume of sputum, and in spite of careful skin protection the excoriation increased. Intravenous fluids and the replacement of pancreatic fluids by mouth and the administration of drugs to inhibit pancreatic secretions did not stop his steady deterioration.

On July 31 the spleen and the pancreas, from its tail to the fibrotic area in the midline, were removed through a transverse incision.

On Aug. 28 his wounds and fistulæ had healed and the lung had re-expanded. Just over a month after his last operation and five months after his injury he was discharged from hospital and has remained well since.

Case 2 (*Figs.* 733-735).—In September, 1952, an officer's wife, aged 42, living in Malaya, began experiencing attacks of pain in the epigastrium and right hypochondrium. These attacks became more severe and frequent until Oct. 11, when she felt so ill that she sent for a doctor.

When first seen she was sitting up in bed holding her abdomen and unable to keep still. There was slight rigidity and guarding of the whole abdomen, with maximum tenderness over the gall-bladder area. The temperature was 100 and the pulse 130.

A diagnosis of biliary colic was made and conservative treatment adopted.

On Oct. 26 she developed an ulcerative stomatitis, and though she continued to have abdominal pain, this had now moved to the left hypochondrium. Her pulse and temperature remained high. The stools became frequent, offensive, and bulky. They contained the ova

of *Ankylostoma duodenale*. Specific treatment was given and the parasites were eliminated.

By Nov. 1 she had developed a cough and radiographs showed a small left-sided pleural effusion. The pain now radiated to the left shoulder. Two days later œdema of the feet and ankles was observed.

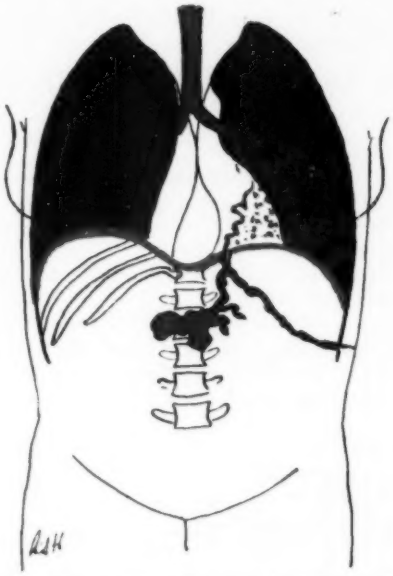

FIG. 733.—*Case* 2. The diaphragm is shown in red. The green shows the area of escaped fluid, the abdominal fistula and the fistula through the diaphragm into the lung.

On Nov. 10 there was a recurrence of severe pain and she had to sit up to get relief. She became cyanosed and breathless and the pulse rose to 130. There was tenderness and œdema of the left loin with a bluish discoloration of the skin.

Left subphrenic abscess was diagnosed and the region was explored by removing the 12th rib. The lesser sac was opened, where multiple areas of fat necrosis were seen and a volume of blood-stained fluid was drained. Only slight improvement followed, but within a day or so the temperature and pulse rose again and there was continuous discharge with marked loss of weight.

On Dec. 3 the original incision was opened again and a small volume of pus evacuated. The discharge of clear fluid remained profuse, but it is noteworthy that there was very little excoriation of the skin.

During the next two months there were recurrent attacks of severe pain, breathlessness, and marked cyanosis. She developed ascites.

On Jan. 23 she was evacuated to the Queen Alexandra Military Hospital, Millbank, by air.

During the flight the fistula closed up and she became very troubled by severe cough and much frothy sputum.

A sinogram outlined the fistula and a deep peri-pancreatic abscess cavity and produced a bronchogram as well. A barium meal recorded the relationship of the cavity to the stomach. A cholecystogram was normal.

The sputum was examined and found to contain pancreatic ferments in strong concentration.

There followed a month of conservative treatment with high-protein diet, the administration of pancreatin and atropine, and continuous suction. There was no improvement and the ascites and œdema increased. Operation was advised.

On March 29 the operation note reads as follows: "Bonney's blue was injected into the fistula and the abdomen opened through a right upper paramedian incision. Eight and a half pints of ascitic fluid were drained from the peritoneal cavity. Strong vascular adhesions of the omentum to the umbilical region were seen and left intact. The liver was engorged but not cirrhotic. The gall-bladder was normal and no calculi were felt. The stomach was closely adherent to the under-surface of the liver. There was marked œdema of the omentum, but the lesser sac was free of fluid. The pancreas was hard and nodular and there were scattered

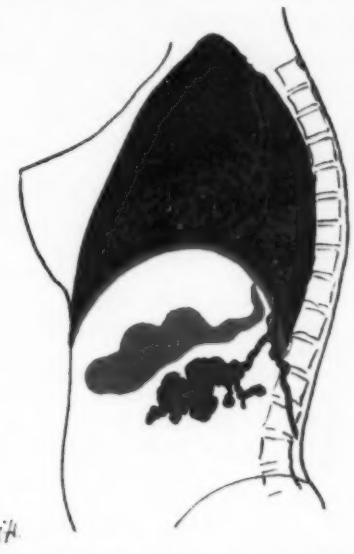

FIG. 734.—*Case* 2. A lateral view showing the relationship of the escaping pancreatic fluid (green) to the stomach and diaphragm (red).

areas of fat necrosis. The posterior wall of the stomach was adherent to the upper border of the pancreas and to the posterior abdominal wall. These adhesions were

FIG. 735.—The under-surface of the diaphragm. The green shaded areas illustrate a possible path of spread behind and between the peritoneum and its folds in relationship to the pancreas, kidney, spleen, and stomach.

separated by blunt dissection and a large cavity containing the Bonney's blue was entered. This cavity ran the full length of the upper border of the pancreas. The widest and most dependent part was in the region of the head. This was drained by inserting a large tube with a catheter threaded through it for irrigation purposes and brought out low in the left hypochondrium.

The day after operation her cough was troublesome and the sputum was blue.

Her convalescence was steady and uninterrupted. Repeated sinograms showed the abscess cavity getting smaller. The large drain was removed gradually and later the catheter. A month after the operation the fistula had healed and two weeks later she was discharged. When seen six months later she had regained 2 st. in weight and was symptom-free.

The lung complications in these 2 cases were their outstanding feature and were alarming. They added difficulties to the planning of their treatment and to the giving of a clear prognosis. Their recovery emphasizes the remarkable reparative powers of lung tissue and the need to "get at the root of the trouble" and deal with the condition below the diaphragm.

Comment.—The points of similarity and difference between the two cases may conveniently be summarized as follows :—

Case 1	*Case* 2
Male, aged 26.	Female, aged 42.
Followed injury.	Followed infection or infestation with *Ankylostoma duodenale.*
(Both were seriously upset by air travel, which seemed to decrease the flow from the abdominal fistula and increase the cough and the volume of sputum.)	
Pain was continuous and wearing.	Pain in severe colicky attacks with breathlessness and cyanosis.
Severe excoriation of the skin.	No excoriation of the skin.
No discoloration of the skin of the abdomen or flank.	Characteristic bluish mottling of the skin around the umbilicus and flank.
(Both had pancreatic ferments in the sputum. Both were emaciated. Both had pain referred to the left shoulder.)	
No œdema of legs or ascites.	Marked œdema of the legs and gross ascites.
(No real improvement with diet, medicine, or replacement of pancreatic secretions in either case. Marked improvement with intravenous saline in both cases.)	
No change in the stools.	Stools bulky and offensive.
Healed after partial pancreatectomy and splenectomy.	Healed after adequate and dependent drainage.
(In each case the bronchial fistula responded to the proper treatment of the abdominal fistula.)	

DISCUSSION

These two remarkable cases are worth recording because they seem unique and because of the unexpectedly successful results.

The serious condition of *Case* 1 precluded any lengthy trial of the more conservative methods of treatment such as continuous suction, dieting, and the giving of drugs to inhibit the pancreatic secretion.

Some operative treatment was indicated, but subsequent events have proved that removal of the damaged portion of the pancreas was all that was needed.

In neither case was it possible at operation to demonstrate the path the fistula followed through the diaphragm. A study of their radiographs and the peritoneal folds below the diaphragm illustrated in *Fig.* 730 tempt me to suggest a spread deep in the folds of the lieno-renal and gastro-splenic ligaments, posterior and lateral to the stomach, and thence through the pleuro-peritoneal canal or even further forward in the left dome of the diaphragm.

E. L. Stern (1930), in his paper on ' Traumatic Injuries to the Pancreas ' states that pancreatic fistulæ never become lined with epithelium and repeatedly become blocked, thus predisposing to chronic recurrent pancreatitis. In his opinion it is better to excise the damaged portion of the pancreas than to try any form of implantation into the upper alimentary tract. *Case* 1 helps to prove his point.

Fortunately in crush injuries such as this one the fistula is likely to follow obstruction to the ducts at the site of compression on the vertebral bodies, and excision of the distal half of the pancreas is comparatively easy.

The adverse effect of air travel in both cases is a point of importance and should be stressed. The distension of bowel and stomach produced at high altitudes in non-pressurized aircraft may compress a thin-walled friable fistula, and prevent drainage, and the increased respiration encourages discharge upwards through any weakness in the diaphragm. If pressurized aircraft or low-altitude flight are not possible it might be wise to leave a drainage-tube in situ.

It is strange that two such apparently very rare cases should have come under my care in so short a time. Is it possible that the condition does in fact occur more frequently but remains undiagnosed ? Their true condition was discovered by chance. In the first case, by associating the increased sputum with the decrease in the discharge from the abdominal fistula, and in the second, by the chance production of a bronchogram when the abdominal fistula was injected with lipiodol. Testing the sputum in future suspected cases may prove their more frequent occurrence.

The use of the sinogram proved well worth while not only as a means of diagnosis but because it gave an indication as to the best method of drainage.

SUMMARY

The case histories of two patients with pancreatico-bronchial fistula are recorded.

Similarities and differences between them are tabulated, and points of importance are stressed.

The methods of treatment are discussed and emphasis is laid on the need to treat the abdominal condition as a means of dealing with the lung complications.

A path of spread through the diaphragm is suggested.

It is postulated that the condition occurs more often than is suspected, but that it remains undiagnosed.

I am grateful to Sir Heneage Ogilvie and Major-General A. G. Harsant for their advice and encouragement, and to the Director-General, Army Medical Services, and Colonel J. E. Snow for permission to publish this report.

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SCLEROSING HÆMANGIOMA OF THE BREAST

BY W. F. WALKER
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SCLEROSING angioma is a pigmented tumour of the skin, rarely recognized as such by the surgeon. In its more pigmented forms it closely resembles a melanoma, and, although a tumour of perfectly benign character, the fact that it is basically an abnormality of blood-vessels and liable to bleed if injured, can add the very touch which seems to indicate the more serious lesion. Its occurrence in the breast further increases the difficulty of the differential diagnosis, and not least in that the surgeon may omit the aid of his dermatological colleagues, in what may well seem an essentially mammary problem.

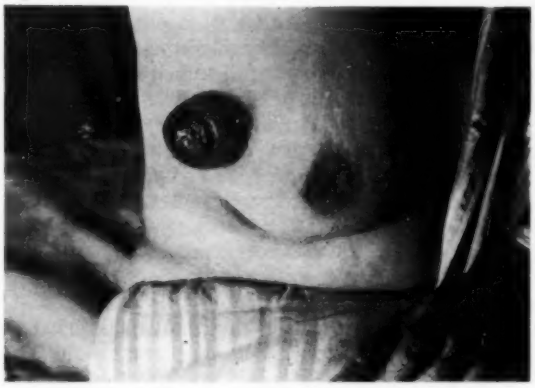

FIG. 736.—Showing the swelling in the right breast.

CASE REPORT

HISTORY.—A married woman of 35 years complained of bleeding from a lump in the right breast (*Fig.* 736). About twenty months before, she had noticed in the breast a bluish discoloured area, quite painless, about the size of a penny (3·3 cm.). It lay flush with the surface of the skin, but eight months later, following a blow, the blue area projected and enlarged to the size of a hen's egg, and ulceration with slight bleeding occurred at the apex. After treatment at home with hot fomentations, the ulcer healed and the swelling subsided, and in about five weeks had disappeared, leaving a blue area similar in appearance to the original.

There was no further trouble until two weeks before admission, when swelling recurred followed by ulceration. Slight bleeding was first noticed about one week after the swelling, and this recurred on the morning of admission.

ON EXAMINATION.—The tumour was a thick columnar mass projecting about 3·75 cm. from the surface of the breast and having a diameter of 5 cm. It had a firm solid wall surrounding a softer or even cystic centre, which was ulcerated and bleeding. There seemed to be no attachment to the deep fascia.

AT OPERATION.—An elliptical skin incision was made around the tumour, which was excised with some of the surrounding breast tissue. It was noted during the operation that the tumour was very vascular (*Fig.* 737).

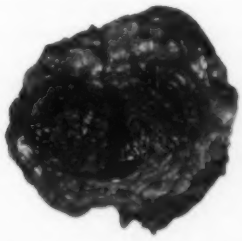

FIG. 737.—Showing the tumour bisected and the brown discoloration suggestive of hæmangioma.

HISTOLOGY (Report by Professor A. C. Lendrum).—This is a fibroxanthoma hæmosiderinoticum (sclerosing hæmangiecton) of somewhat cellular type. The superficial parts of the tumour, which are separated from the overlying epithelium by a fairly dense layer of fibrous dermis (*Fig.* 738), are cavernous and change below rather suddenly into the characteristic tangled whorled hæmosiderin-laid fibrous structure of the sclerosing angioma. The changes of fibrocystic disease are present in the mammary tissue.

DISCUSSION

Sclerosing angioma is not a common tumour and only in recent years has the condition been recognized as such. In a series of dermatological lesions occurring in the American Armed Forces, Allen (1948) found 426 cases of sclerosing hæmangioma in 8000 skin lesions, of which 6475 were neoplasms of skin. This is an incidence of about 7 per cent of skin tumours. The sites of the tumours were not specified. Although the tumour is sometimes obviously hæmangiomatous, it is by no means always so, even to the microscopist, and it is therefore perhaps not surprising that in various writings on

hæmangioma occurring in the breast the sclerosing form of the tumour is not even mentioned (Menville, 1933; Geschickter, 1947). On the other hand, in a series of 67 histologically proven examples of sclerosing angioma (Gross and Wolbach, 1934), 3 had come from the breast. Other workers, interested

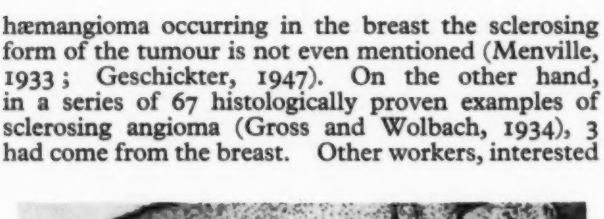

FIG. 738.—Under the thickened epithelium is a fibrous layer of dermis and then the tumour with its ill-defined edge. The cavernous venous channels are present only in this superficial layer of the tumour. H. and E. (× 37·5.)

in this tumour rather than the site, have not had this incidence in the breast; thus Dawson (1948) in 14 cases had no mammary ones; Ewing and Powell (1951), had 9 all extramammary, and Dr. John Milne

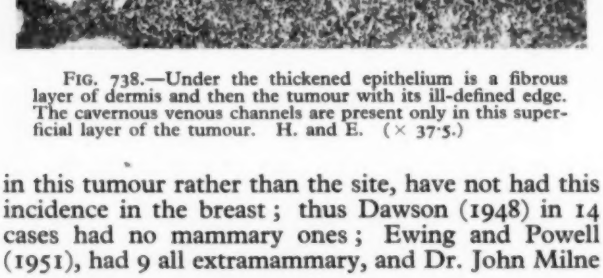

FIG. 740.—This shows scattered clumps of pale lipophages among the heavily pigmented cells, and two larger aggregates with the acicular clefts characteristic of breakdown lipoid as seen in paraffin sections. Celestin blue and chlorantine red. (× 45.)

of Glasgow University has been kind enough to tell me that of the 20 examples he has studied none were from the breast.

The sclerosing process which characterizes these hæmangiomas is described by Gross and Wolbach (1934) as an overgrowth of the tumour stroma, with concentrically arranged fibroblasts encircling and constricting individual capillaries, the contraction of this newly formed connective tissue compressing the capillaries gradually and concentrically (*Fig.* 739).

Two other features of histological interest characterize this condition of sclerosing angioma. Firstly, there is the occurrence within the stroma of the tumour of abundant hæmosiderin, which may be present in sufficient quantity to give the cut surface a dark brown or even black colour. In

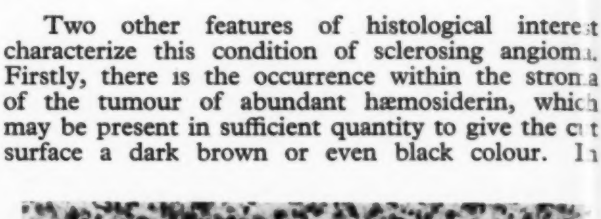

FIG. 739.—This field from one of the less pigmented zones shows the appearance of interrupted whorling typical of the sclerosing angioma. H. and E. (× 140.)

routine H. and E. sections of such tumours, the pigment is usually found easily and Perl's reaction may reveal a surprising amount of prussian blue, mostly contained within phagocytes in the stroma and particularly around the periphery of the tumour. Some of the phagocytes are clearly recognizable as individual cells packed with granules, but in other places the hæmosiderin granules are seen as twig-like distorted rows which are presumably contained within cells compressed by the connective tissue and resembling the curiously twisted and extended polymorphs seen in the inflamed cornea.

Iron-reacting crystals and crystalline encrustation of elastica and reticulum such as occur in the region of hæmosiderin deposits in the spleen of the case with splenic anæmia or in the lung of a patient with cardiac disease (Lendrum, 1950) have not been seen in the present specimen or in a number of other examples of this type of tumour (Lendrum, 1953). In the lung, encrustation by iron of the reticulum and elastica leads to fragmentation of these structures, and only thereafter to the reactive changes, viz., accumulation round the fragments of syncytial giant-cell masses, even to the extent of forming zones of giant-cell granuloma, and then fibrosis. But fibrotic proliferation of the stroma also occurs in the pulmonary alveolar walls around the siderophage collections without there being any sign of previous encrustation, fragmentation, or giant-cell formation; in this, presumably, there is a direct stimulation of the fibrogenic activity (Lendrum, 1950). In a similar manner, stromal proliferation can occur in the region around a basal traumatic hæmatoma of the subcutaneous tissues, and this may even become neoplastic, invading the surrounding tissues, as a sarcoma (Pack and Anglem, 1940). In both these latter types of proliferation it is now suggested (Lendrum, 1953) that the stimulus to proliferation

may be not the iron from the extravasated and broken-down erythrocytes, but rather the lipoid or fatty substances released from the erythrocytes. The possible fibrogenic activity of lipoid or other fatty substances, within the lung at least, is suggested by the observations of Morgan on intrinsic lipoid pneumonia (1952). Such a hypothesis is possibly relevant to the sclerosing angioma, because the other histological feature of this tumour is, in fact, the presence of fatty materials.

In paraffin sections of sclerosing angioma it is common to find foam cells—lipophages—occasionally in very large numbers (*Fig.* 740). Sometimes these cells are multinucleated and resemble the Touton cells of dermal xanthomata. In frozen sections the fat in these cells not only stains strongly with Sudan red, but is also largely doubly refractive. Indeed by polarized light there appears to be a considerable quantity of fine crystalline and amorphous material which is faintly or not at all stained with Sudan red. Application of the Schultz reaction to a frozen section of one of these tumours gave an immediate and strong reaction for cholesterol, confined to the zones where the sudanophil material was present.

The source of the fat in these tumours is attributed by Gross and Wolbach (1934) to nutritional causes, because of the lack of evidence of any metabolic disturbance. But in view of the gross evidence of previous hæmorrhage, of the fact that the lipophages are seen in the regions where siderophages are abundant, and of the large content of sterol, it seems reasonable to assume that the fatty material has come from the extravasated erythrocytes.

Some examples of this type of tumour show practically no iron or fat, and we are, therefore, faced with the problem whether such cases exemplify the late stage of a lesion from which the deposits have been removed by phagocytic scavenging, or could they be the early stages of a curious and fibrotic lesion which is characterized in its later phases by massive extravasation of erythrocytes? The histological structure of such iron-free examples is on the whole not one to suggest a special liability to extravasation, and on general principles this hypothesis seems less acceptable than the idea that the hæmorrhage precedes the fibrosis.

The argument thus comes to the problem of why an angioma should show this extraordinary liability to hæmorrhage. This might be due to trauma, but large cavernous hæmangiomata occur in the skin without any suggestion of comparable sclerotic change. Another curious fact is that the sclerosing angioma seems rarely, if ever, to have shown the characteristic clinical picture of Wood's 'painful subcutaneous tubercle'.

DIAGNOSIS

In the present case the probable diagnosis seemed to be a tumour of mammary gland, and the possibility of a primary dermal lesion was rather too readily rejected, although enough uncertainty remained to encourage us to undertake a local removal. In sites other than the breast, the surgeon is less likely to have his mind led away from skin, and the main difficulty in diagnosis is then to distinguish this tumour from melanoma, a difficulty which Roussy, Huguenin, and Oueyn (1935) regard as being clinically insoluble. The seriousness of this difficulty is well brought out by Dawson (1948) and by Ewing and Powell in their striking series (1951).

If the surgeon is aware of the difficulty and realizes that this clinical picture may mean either a completely benign or a dangerously malignant neoplasm, the correct approach would seem to be consultation with the dermatologist and the pathologist. The latter might well be able, on the basis of an immediate frozen section, to state the nature of the lesion and thus indicate whether the operation should proceed to a removal of the related lymphatics. If a pathologist is not available the cut surface of the removed tumour could be treated with a mixture of equal parts of 4 per cent hydrochloric acid and 4 per cent potassium ferrocyanide. Fresh reagents are unlikely to give a false negative result and if a pigmented tumour fails to show a strong bright blue colour the tumour is almost certainly a melanoma.

SUMMARY

The occurrence of a pigmented sclerosing angioma within the skin of the breast presented a difficult diagnostic problem.

This tumour although completely benign, can exactly mimic a melanoma.

If this point is appreciated arrangements can be made to obtain an accurate diagnosis at operation.

A few points in the pathology of sclerosing angioma are discussed.

I wish to record my thanks to Professor D. M. Douglas for his permission to publish this case. I should also like to thank Professor A. C. Lendrum for his invaluable advice in the preparation of the paper, and also to Dr. H. G. Morgan who prepared the microscopical specimens.

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GIANT FIBRO-ADENOMA OF THE BREAST: CYSTOSARCOMA PHYLLODES

By MAURICE SILVERSTONE, Liverpool

Cystosarcoma phyllodes is an uncommon lesion of the breast. Lee and Pack (1931, b), from the New York Hospital for Cancer and Allied Diseases, collected 109 cases from the literature including 4 of their own cases, and added 2 further cases of their own. Owens and Adams (1941), in reporting a case, found records of at least 12 other cases omitted from the list collected by Lee and Pack (1931, b). McDonald and Harrington (1950), from the files of the Mayo Clinic for the period 1904 to 1943, found 13 cases of giant fibro-adenoma. They found records of a further 11 cases not included in the two previous articles. Hill and Stout (1942), in recording 15 cases from the Columbia University College of Physicians and Surgeons seen between 1911 and 1940, noted at the same time that there were 1990 cases of carcinoma, 11 of varying types of sarcoma, and 2 of leukæmic tumour. The cases of cystosarcoma phyllodes represented 2·1 per cent of all tumours removed with the diagnosis of fibro-adenoma. Treves and Sunderland (1951), mainly from the New York Memorial Centre for Cancer and Skin Diseases, reported 77 cases, of which 10 were recurrences which had had their original operation performed elsewhere. Stephenson, Gross, Gumport, and Meyer (1952) reported 15 cases, of which 13 were seen between 1939 and 1950 at the University Hospital of the New York University. Geschickter (1945) in 6044 breast cases had 2499 carcinomas of the female breast, 30 of the male breast, 62 cases of Paget's disease, 62 sarcomas, 600 fibro-adenomas, and 58 cases of giant myxoma (cystosarcoma phyllodes). Willis (1948) in fourteen years had 550 carcinomas of the female breast, 2 of the male breast, 6 cases of Paget's disease, 1 sarcoma, 15 benign papillomas, and 130 fibro-adenomas, of which 3 were giant fibro-adenomas. Altogether some 250 cases have been reported.

Cystosarcoma phyllodes was described in some detail by Müller (1838) as follows:—

"The tumour forms a large mass with a more or less uneven surface. The fibrous substance which constitutes the greater part of it is of a greyish white colour, extremely hard and as firm as fibro-cartilage. Large portions of the tumour are made up entirely of this mass, but in some parts are cavities or clefts not lined with a distinct membrane. These cavities contain but little fluid; for either their parietes which are hard like fibro-cartilage and finely polished lie in close apposition with each other, or a number of firm irregular laminæ sprout from the mass and form the wall of the fissures, or excrescences of a foliated or wartlike form sprout from the bottom of the cavities and fill up their interior. These excrescences are perfectly smooth on the surface and never contain cysts or cells. The laminæ lie very irregularly and project into the cavities and fissures like the folds of the psalterium in the interior of the third stomach of ruminant animals. In one instance the author saw these laminæ here and there regularly notched or crenated like a cock's comb. Sometimes the laminæ are but small and the warty excrescences from the cysts very large, while in other instances both are greatly developed. Occasionally these warty excrescences are broad, sessile, and much indented; others have a more slender base and somewhat resemble cauliflower condylomata.

Tumours of this kind attain an enormous size, hitherto the author has seen them only in the female breast nor are they even there of frequent occurrence. They are decidedly innocent, occur earlier than is usual for cancer in the mamma to develop itself, and sometimes they appear even in youth: they have but little tendency to grow to the skin or to the adjacent muscles and are not attended with retraction of the nipple. They are not disposed to soften internally, but continue to grow slowly until they have attained an enormous size when they at length burst and a very ill-looking suppurating fungus forms upon their surface. Even in this state, however, operation has been performed with a successful result. Swelling of the axillary glands is not a common occurrence and when it is met with is the consequence of simple irritation and subsides after the operation. The extraordinary forms which cystosarcoma phyllodes assumes at once suggest the notion of its cancerous nature, and yet the disease is perfectly innocent and as far removed from carcinomas as are those non-suppurating cauliflower condylomata of the penis and of the female genitals which have so often been mistaken for cancerous structures."

Similar tumours had previously been described by Chelius (1828) under the term 'cystic hydatid' and by Cumin (1827) and Astley Cooper (1829) as 'cellular hydatid'. Since Müller's description, the tumour has suffered from a plethora of different names, of which the following are some: Cystosarcoma (Fischer, 1835), hydatid tumour of the breast (Warren, 1839), serocystic tumour (Brodie, 1846), cystic sarcoma (Erichsen, 1852), telangiectatic cystosarcoma (Amado, 1872), proliferous cystosarcoma (Gross, 1874), intracanalicular papillary fibromyxoma (Bigelow, 1876), intracanalicular sarcoma (Iversen, 1877), intracanalicular myxofibroma (Watson, 1878), sarcoma with lacunar cysts (Raingeard, 1882), intracanalicular myxoma (Jüngst, 1884), proliferating cystic tumour (Leser, 1888), cystosarcoma myxomatoides (Schmidt, 1889), cystosarcoma mammæ proliferum (Noetzel, 1892), cystosarcoma papillare (Pilliet, 1893), papilloma intracanaliculare (Kursteiner, 1894), intracanalicular fibroma (Pearce, 1896), intracystic mammary sarcoma (Snow, 1899), cystofibrosarcoma (Grohé, 1900), pearly cystosarcoma phyllodes (Beneke, 1902), adenoma pseudosarcomatodes (Beneke, 1902), fibro-adenoma intracanaliculare papillare (Schmuckert, 1904), intracanalicular fibroma with sarcomatous change (Friend, 1909), adenomyxofibroma intracanaliculare papillare (Gorham, 1911), fibrolipo-adenoma intracanaliculare sarcomatodes xanthomatodes mammæ (Binkert, 1924), giant intracanalicular myxoma (Lee and Pack, 1931, a), giant intracanalicular fibro-adenomyxoma (Lee and Pack, 1931, b), giant intracanalicular fibro-adenoma (Owens and Adams, 1941), adenofibrosarcoma (Hill and Stout, 1942), giant fibro-adenoma (McDonald and Harrington, 1950), cystosarcomatoid phyllodes (Horton and Baker, 1950).

The multiplicity of these names indicates the differing emphasis laid by individual authors on the varying structure either macroscopical or microscopical and has led to some confusion both in the recording of cases and the classification of the condition into a definite pathological group. The lesion described by most authors is a massive tumour of the breast, yet Hill and Stout (1942) include in their series their smallest tumour of 3·5 cm. diameter and their largest, which weighed 14 lb. (6·4 kg.). Treves and Sunderland (1951) would also classify the tumour on a basis of microscopical pathology and in their series they include a lesion 1 cm. in diameter. They would exclude merely large fibro-adenomata if their histology did not conform to that of cystosarcoma phyllodes. McDonald and Harrington (1950), on the other hand, would only include such lesions as involved the major portion of the breast (more than four-fifths) and weighed more than 500 g. The innocence of the tumour stressed by Müller and other authors is generally recognized, yet in 91 cases of Lee and Pack's (1931, b) collected series in which the outcome was noted, 5 recurred, 3 following simple mastectomy, and 1 each after simple excision and after radical mastectomy. In an additional case dealt with by simple mastectomy, pulmonary metastases occurred. White (1940), in reporting a case which recurred locally and developed metastases in the other breast, mediastinum, and lung, pointed out that sarcomatous change in cystosarcoma phyllodes had been reported by Friend (1909), Westermark (1918), and Silvan (1919). Prym (1912) and Steckler and Landman (1950) reported metastases to the lung, and Pierson (1950) to the chest wall and ischium and ilium. Cooper and Ackerman (1943) reported a case with metastases in the axillary glands, and Treves and Sunderland (1951) in their series of 77 cases, of which 18 were malignant, had also 1 case with axillary metastases. They pointed out that spread is usually blood-borne, however, the lungs being the most frequent site for secondaries. They felt that the term 'cystosarcoma phyllodes' should be qualified by the addition of the words innocent or malignant. McDonald and Harrington (1950), while agreeing that cystosarcoma phyllodes was an innocent fibro-adenoma in 90 per cent of cases and a fibrosarcoma in the remainder, felt that the term should be avoided and tumours classified either as giant fibro-adenomas or fibrosarcomas. In their series fibro-adenomas which showed malignant changes in the stroma were excluded.

CASE REPORTS

Case 1.—E. G., a married woman, a nullipara 65 years of age, was referred to me by Dr. Grierson on March 7, 1941 with a swelling of the breast. The patient had noticed the swelling in her breast since the previous June and it had grown to its present size in that time. It was painless and the chief complaint was of its size. There was no previous trouble in her breast nor any relevant details in her personal or family history. Examination showed a mass in the inner half of the right breast 8 cm. by 5 cm. This was firm, but there were associated two fluctuating areas. It was apparently adherent to skin and there was some *peau d'orange*. Axillary glands were not palpable. The diagnosis of serocystic disease was considered, but in view of the apparent adherence to skin and the presence of fluctuating areas the possibility of a low-grade inflammatory lesion such as tuberculosis was also entertained. Aspiration of the fluid and examination showed blood-cells, an occasional pus cell, but no organisms. Culture of the fluid was sterile. Arrangements were made for her admission for operation, but she did not come in when sent for. This was due to the illness of her husband. Subsequent to his death, with the increasing size of the tumour, she took to her bed, where she stayed for six months. During

FIG. 741.—*Case* 1. Photomicrograph showing highly cellular stroma. (× 21.)

this time she applied a herbal ointment (Firth's green salve), which she thought had produced ulceration of the skin and a watery discharge. No blood came away, but she was convinced that the loss of the water was beneficial and her breast would improve. Finally she was persuaded by a doctor friend to consult her own doctor, who referred her back to me in the hope that I would deal with the fungating mass.

She was admitted on June 16, 1942. At that time the inner two-thirds of the right breast was occupied by growth which had fungated through the skin and looked a most malignant and unpleasant lesion. It was evident, however, that with the absence of glands, the non-invasion of skin, the size of the tumour, its progress, the excellent general condition of the patient that the diagnosis was serocystic disease of the breast or rapidly growing soft fibro-adenoma. A simple mastectomy was performed and convalescence was uneventful. The pathological report on the tumour unfortunately was that it was a spindle-cell sarcoma—due to the misinterpretation of the highly cellular stroma—and so though clinically it was a benign lesion she was referred for post-operative X-ray therapy. (*Fig.* 741.) This was given to two areas, right breast and axilla, 20 × 20 cm., and right supraclavicular region, 15 × 15 cm. The total dose to each area was 1851 r, given twice weekly from Oct. 29 to Nov. 24. When last seen on Jan. 20, 1953, there was no evidence of any recurrence and she was very fit, having had no trouble referable to her breast. Since irradiation is ineffective in cystosarcoma phyllodes, the result in this patient confirmed the clinical diagnosis originally put forward of a benign cystosarcoma phyllodes.

Case 2.—M. M., an unmarried woman, 36 years of age, was seen in consultation with the late Dr. Morgan. She had noticed an increase in size of her left breast for four to five years, but this increase had been most rapid in the past twelve months. There was no pain, but there had been increasing discomfort from the weight of the breast. She was a very shy woman, and the family had only noticed the deformity in her chest region lately. They had had the greatest difficulty in persuading her to see a doctor. On close questioning and because

the tumour was so large, the mother thought she had noticed her daughter's unilateral breast prominence since the previous Christmas. There was nothing relevant in her previous history. The left breast was three to four times the size of the right, the lowest portion being

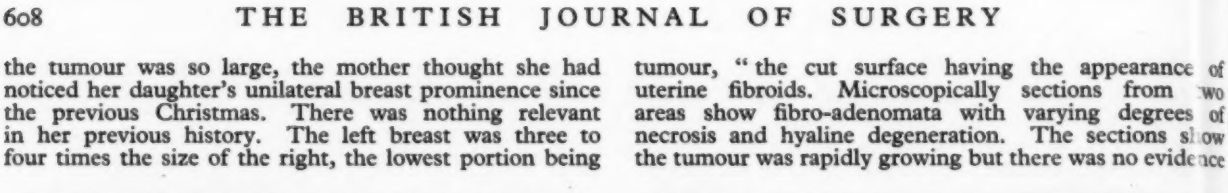

FIG. 742.—*Case* 2. The size of the breast.

FIG. 743.—*Case* 2. Macroscopical appearance of the tumour

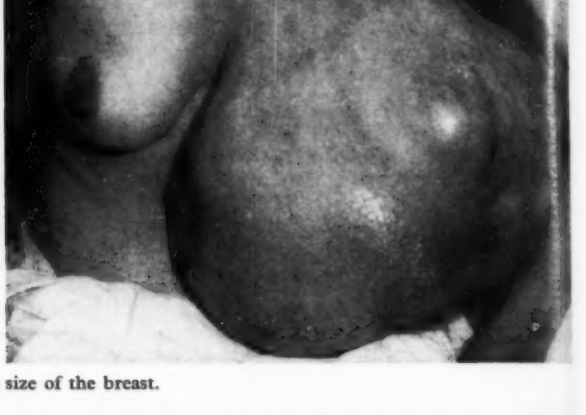

FIG. 744.—*Case* 2. Microscopical appearance of the tumour. (× 27.)

below the umbilicus, and when she sat up almost rested on her thigh. The tumour was firm, bosselated, the skin stretched and purple in parts, with dilated veins. In the dependent part of the breast *peau d'orange* was present. Axillary glands were not palpable. The general appearance and size of the tumour, about 30 × 25 × 20 cm., can be judged from the photograph (*Fig.* 742.)

She was admitted to hospital within a week, on Sept. 25, 1952. Pre-operative investigation showed hæmoglobin 51 per cent, with R.B.C. 3,600,000, Group B Rh negative. Operation was performed on Sept. 27. A simple mastectomy was carried out, with removal of the thinned-out purple skin, which was closely associated with the most prominent part of the tumour. The tumour was œdematous or hydropic and vascular. The perforating branches from the chest wall were the size of a lead pencil. Some of the superficial fibres of the pectoralis major were also removed, not because it was thought that infiltration had taken place, but because œdema was present here. Immediate skin-graft was applied to the bare area. During and after operation, 2 pints of blood were transfused. Convalescence was uneventful and she was discharged from hospital on Oct. 16. The weight of the tumour was 15 lb. (6925 g.). *Fig.* 743 shows the macroscopical appearance of the tumour, "the cut surface having the appearance of uterine fibroids. Microscopically sections from two areas show fibro-adenomata with varying degrees of necrosis and hyaline degeneration. The sections show the tumour was rapidly growing but there was no evidence of malignant change in the portions examined" (Dr. R. M. Rawcliffe). (*Fig.* 744.) The patient, seen in February, 1953, was very well, with no evidence of any recurrence.

DISCUSSION

Aetiology.—Apart from 3 cases in males included in Lee and Pack's (1931, b) collected series, all the reported cases have been in women. They have been seen at all ages beyond puberty—McDonald and Harrington (1950) reported 2 bilateral cases in girls of 13—the average age being 45. They occur a little more frequently in married women than in single. Lee and Pack (1931, b) considered lactation and nursing difficulties to be a factor in the metamorphosis of a simple fibro-adenoma to cystosarcoma phyllodes, but Treves and Sunderland (1951) found no such association in their cases, and of the two cases reported here, one single and one married, neither had had children. Lee and Pack stressed the development of the lesion from a pre-existing fibro-adenoma, usually of the intracanalicular type, but occasionally of the pericanalicular type. This

was found in 47 of their 111 cases, with an average duration of 7 years. There was evidence of a pre-existing fibro-adenoma in 7 of the 15 cases reported by Stephenson and others (1952), with an average duration of 9·7 years, but in only 2 of the 13 cases reported by McDonald and Harrington (1950). In the series reported by Treves and Sunderland (1951) 53·2 per cent grew uniformly slowly, 26 per cent grew rapidly after an initial slow growth, 13 per cent grew rapidly from the first, and in 7·8 per cent no details were available. They thought they recognized areas of pre-existing fibro-adenoma in 21 of their 77 cases, but felt that since 54 per cent of their cases had a history of less than a year these were not likely to have been preceded by a fibro-adenoma.

Pathology.—Macroscopically the lesion is as described by Müller (1838). The tumour is surrounded by a capsule which may ramify into its substance, forming indefinite lobules. The surface is uneven and irregular, with variable but usually firm consistency, unless extensive necrosis has supervened. The cut surface is bulging, greyish-white or pinkish-grey in colour, whorled and elastic. In the intracanalicular type the growth is traversed by clefts into which polypoid masses may project so that these clefts have a branched or stellate appearance. Cysts if present are of two types, (1) those resulting from continued secretion of the epithelial cells and lined by a smooth glistening membrane, and (2) those produced by necrosis and liquefaction (Hill and Stout, 1942). The pericanalicular type is usually smaller, firmer and seldom cystic. Microscopically Lee and Pack (1931, a, b) stressed the myxomatous stroma with cellular pseudosarcomatous regions. The picture is of a fibro-adenoma with relative overgrowth of the stroma demonstrable in increased cellularity, increased amount, or both. In the firmer areas the stroma is cellular, composed of spindle-shaped fibroblasts and fibrocytes. In other softer areas the cells are fewer in number and the collagen fibres form a loose network. In the soft gelatinous areas found usually in the vicinity of the epithelial lined clefts the stroma was more mucoid or œdematous. Lee and Pack pointed out that the metaplasia of the stroma into such myxomatous tissue was the characteristic feature of the intracanalicular variety. The connective-tissue cells usually are regular, though a slight degree of cellular pleomorphism is evident occasionally. Giant cells may be seen. Mitoses are rare. In the more malignant type mitoses are much more frequent and pleomorphism more marked. The epithelial element is scarce or prominent, but hyperplasia is infrequent. Squamous metaplasia sometimes occurs and occasionally epithelial pearls are formed. Where malignancy occurs this is almost invariably in the connective tissue element and the epithelial elements diminish and may be scarcely recognizable.

Clinical Features.—These are fairly characteristic. Occasionally in a patient with a slow growing fibro-adenoma which has been present for years rapid growth takes place. In others the history is of rapid growth from the beginning. Usually in one breast, rarely both are involved. The tumour is massive as a rule, smooth, or nodular, with areas of softening or fluctuation in the bigger lesions. The overlying skin is tense, with enlarged veins coursing over it, and ultimately the skin may ulcerate and the tumour may fungate through. There is no fixation to skin or deeper structures, and after ulceration it is possible to pass a probe between the skin and the tumour. In both the cases reported *peau d'orange* was present. Glands are not involved except secondary to infection. There may be a serous discharge. The condition of the patient remains good, the chief complaint being the weight of the tumour. It is likely that in future with the earlier attendance of patients for treatment the massive tumours will no longer be seen. This picture is readily recognizable and the diagnosis from breast abscess, simple cysts, tuberculosis, fibro-adenoma, and carcinoma is relatively easy. From diffuse hypertrophy of the breast, especially in the bilateral cases and in younger patients, diagnosis may be more difficult, but careful palpation will reveal a relative absence of irregularity in this condition. From sarcoma the diagnosis may be difficult but the fixation of the former is the important sign as well as invasion of skin.

The diagnosis of the lesion clinically can therefore usually be made if it is borne in mind, and this is of considerable importance to the patient as it lends itself to simple and satisfactory treatment. The pathologist, on the other hand, diagnoses the lesion on the basis of an increased stroma and with myxomatous change, and finds lesions which are small and not clinically resembling the massive tumour described. The clinician is not likely to diagnose these cases as cystosarcoma phyllodes. It is important to include both the large and the small lesions in the one term because of the possibility of local recurrence after treatment and of distant metastases. It is difficult to assess the frequency of this. In Lee and Pack's collected series, there were 5 local recurrences and 1 with distant metastases. Treves and Sunderland, on a pathological diagnosis of cystosarcoma phyllodes, had 18 malignant and 18 borderline cases out of 77 tumours. They felt, however, that this proportion was likely to be greater than the true one because recurrences had been referred to their hospital from other units. In any series, however, are found cases which clinically and pathologically are classified as benign and yet are followed by the appearance of recurrence locally and occasionally by distant metastases.

Treatment.—Simple mastectomy is the treatment of choice. Local excision can be done if the lesion is small enough, but if recurrence takes place this should be followed by simple mastectomy. The lesion is usually benign, but if clinically there is any local invasion of muscle or skin a radical mastectomy should be carried out. X-ray therapy has been used for distant metastases, but is of doubtful value.

SUMMARY

1. Two cases of giant fibro-adenoma are reported.

2. The benignity of the tumour is noted, but the possibility of the lesion being malignant or taking on malignant characters is stressed; this in the absence of any definite change in the clinical or pathological character of the primary tumour.

3. Treatment by simple mastectomy is advocated.

I am indebted to Dr. R. M. Rawcliffe for the pathological report on the second case, the Pathological Department of the University of Liverpool for *Figs.* 741 and 743, and Mr. Lee, of the Central Photographic Department of the University, for *Figs.* 742, 744.

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PIGMENTED CONGENITAL EPULIDES OF NEURO-EPITHELIAL ORIGIN

THE DESCRIPTION OF A FURTHER CASE WITH SUCCESSFUL EXCISION AND SURVIVAL

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THE term 'epulis', according to Dorland (*American Illustrated Medical Dictionary*), means no more than a tumour on the gum (*ἐπί* and *οὖλον*). In adults, the term is mainly restricted to fibrous formations and to giant-cell tumours of the bone. In young infants a congenital form of epulis is accepted as a rare clinical form, and though its nature and genesis are still debatable, it is now generally described as a granular-cell 'myoblastoma'.

This paper describes a still rarer variety of congenital epulis, of which only 6 cases have been reported. It differs from the granular-cell type in being of neural structure, with melanin pigmentation, and in our opinion is derived from retinal rudiments. It is the first example of this type to be recognized in British literature, although, as we shall discuss later, such growths have been described under other names.

CASE REPORT

A male infant, two months old, was admitted to the Royal Hospital for Sick Children, Glasgow, in November, 1950, with a history of swelling of the upper gum, first noticed when he was one month old. On examination the alveolus projected forwards on the left of the midline and the hard palate showed a smooth rounded swelling which was considered to be a bone cyst occupying and expanding the alveolus at the junction of the premaxilla and the maxilla (*Fig.* 745).

This cyst was exposed by a horizontal incision in the buccal sulcus reflecting the mucous membrane, and was shelled out of its bony bed intact, save at the upper and medial limit where the dark chocolate-coloured tissue which filled the cyst continued upwards towards the nasal sinuses. At the periphery of the bed the bone edge was flared outwards, but was otherwise healthy. The impression at operation was that the cyst, though growing more quickly than the child, was essentially

simple in character, possibly a dermoid, and the contents suggested altered hæmorrhage.

The biopsy tissue sent for histological examination was a fleshy tumour mass, approximately 2 cm. cubed,

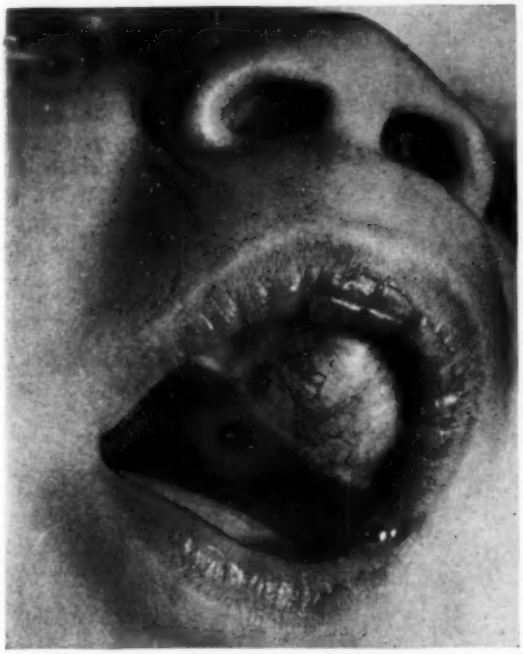

FIG. 745.—Photograph of the child, aged 2 months, when first examined to show the position of the tumour. (× 1·6.)

FIG. 747.—Photograph of the child nineteen months after the first operation to show the satisfactory repair of the alveolus. (× 1·6.)

with a well-formed incisor tooth on the surface. Histologically the general appearance was as in *Fig.* 746. The main feature was the dark staining cells in solid masses defined by loose fibro-vascular strands. In the centre of a few of these masses larger clear cells were found. The possibility of an odontoma was considered, and though long strips of pigmented columnar cells were observed, their significance was initially misunderstood, especially as they appeared to react positively to Von

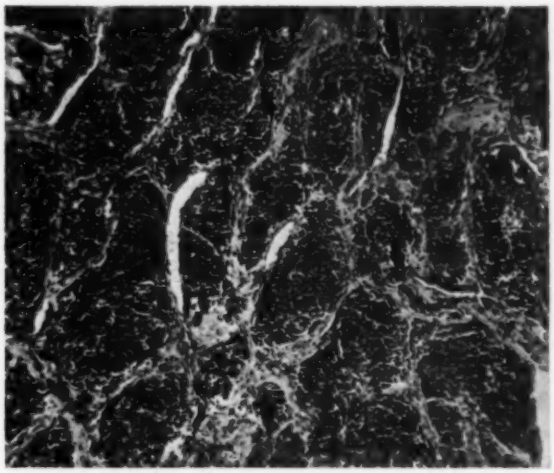

FIG. 746.—Section from first biopsy to show solid masses of dark staining cells. H. and E. (× 45.)

Kossa's silver stain for calcium. The tumour was considered to be benign.

Healing of the wound was satisfactory, but recurrence of the swelling was noted about three weeks later. The question of malignancy now arose. The radiotherapist was unwilling to treat the tumour on account of probable subsequent interference with the growth of the facial bones. The growth continued to increase slowly in size and it was decided to attempt a more radical local excision.

FIG. 748.—The naked-eye appearance of the tumour to show the pigmentation. (× 1·6.)

This was done in February, 1951, the child being now five months old. There was no longer a cyst but solid dark tissue which seemed to be derived from tooth germs. The alveolus was curetted; seven unerrupted teeth were removed and the bony hollows left bare were seen not

to be infiltrated by the tumour which extended back to the soft palate. After curetting this area a cavity which remained seemed to be free from tumour, though distorted. The hard palate in particular bulged downwards very markedly. The cavity was packed with alginate wool and the mucoperiosteum stitched over this.

The amount of blood lost (7 oz.) was replaced by transfusion and the child made a good recovery, the wound healing well. The distorted bone of the alveolus and palate reformed their normal contour and in August, 1952, when the child was twenty-three months old, there was no trace of any aberration save for the absence of some upper teeth (*Fig.* 747).

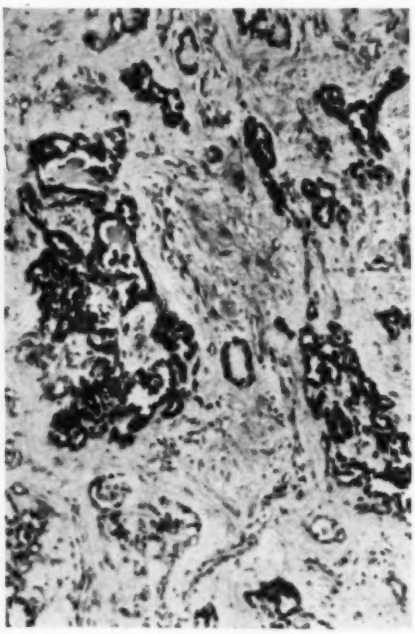

FIG. 749.—General view of the area composed of primitive tubules lined by heavily pigmented cubical cells. H. and E. (× 60.)

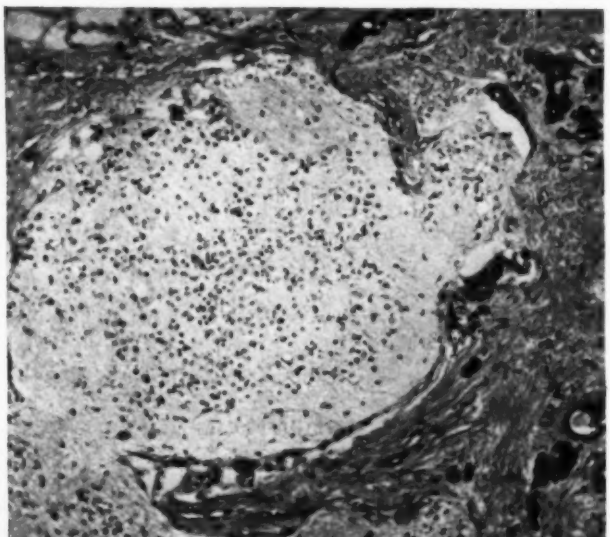

FIG. 751.—Area of well-defined glial tissue. H. and E. (× 60.)

From this second operation a much larger quantity of tissue was available together with several pieces of bone from the alveolus. The tissue was remarkable for its dark colour (*Fig.* 748). Several blocks were cut to illustrate the various structures involved in the tumour.

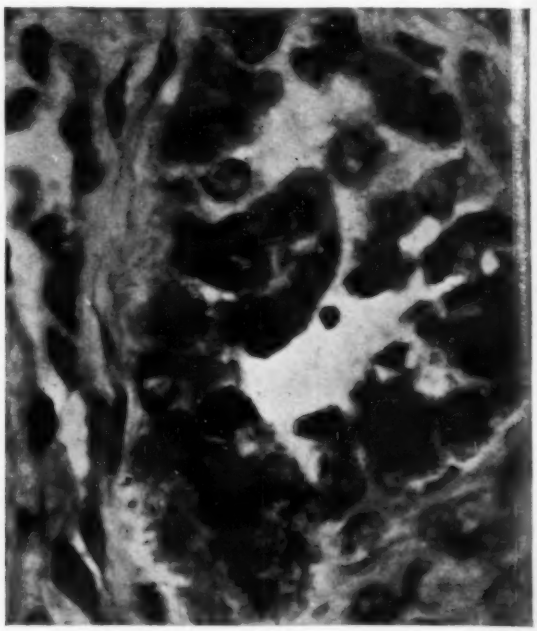

FIG. 750.—High-power view of a pigmented tubule where the pigment is comparatively thin. The bacillary appearance of the pigment may be seen. H. and E. (× 460.)

HISTOLOGICAL FINDINGS.—Histologically, the growth could be divided broadly into three structural types. The first was the solid alveolar pattern as seen in the first biopsy (*Fig.* 746). The second was composed of clefts of primitive tubules lined by heavily-pigmented cubical cells in a moderately cellular connective-tissue matrix (*Figs.* 749, 750), well supplied by capillaries; no necrosis was found in any of the many blocks examined. The third type was composed of small defined areas of glial tissue (*Fig.* 751). These provided the key to the essential nature of the tumour. Further examination of other sections led to the recognition of the cells in the solid masses as neuroblasts; stages between these cells and their full differentiation into glial cells could be seen fairly often (*Fig.* 752). Many of the cells in this differentiated part had the boxed appearance of oligodendroglia (*Fig.* 752), and in this same field differentiation to unipolar spongioblasts was seen in the upper part (X) of the glial tissue, with a peripheral zone showing transitions between the two types. In many places the pigmented cubical cells lining the clefts were in direct continuity with masses of neuroblasts (*Fig.* 753). This indicated differentiation towards primitive retina, an opinion further supported by the bacillary morphology of the pigment granules in the cubical cells. The close proximity of capillaries to the neuroblastic tumour elements ensured a good blood-supply and explains the absence of necrosis. Examination of many blocks of tissue failed to reveal neoplastic derivatives of other germ layers, i.e., there is no histological evidence that the growth is a teratoma.

DISCUSSION

Detailed examination of the histology of this tumour leaves no doubt about its neural origin. Halpert and Patzer (1947) published the case of a

six-months-old female with a maxillary tumour of retinal origin exactly similar to the one we describe. The site, the dark mass, the histological and X-ray appearances are almost identical in the two cases. Halpert and Patzer claim their tumour as the first of its kind *recorded*, and though the claim is acknowledged by Clarke and Parsons (1951) and by Martin and Foote (1951) when recording their own single cases, it would be more accurate to say *recognized*, because it is clear that similar lesions have been mistakenly described by previous writers as pigmented adamantinomata. Our case would therefore appear to be the fourth example in an infant of this melanotic congenital epulis recognized as neural in origin. There are in fact only two records of so-called pigmented adamantinomata. Wass (1948) described an example of this tumour in a female child aged five months in the lower jaw, and Battle, Howell, and Spencer, (1952) recorded one in a six-weeks-old male ; in both of these the histology of the tumour is similar to ours, but the site is in the lower jaw at the right side. This curious diagnosis led us to examine the literature on tumours classified as ameloblastoma, adamantinoma, etc. Churchill

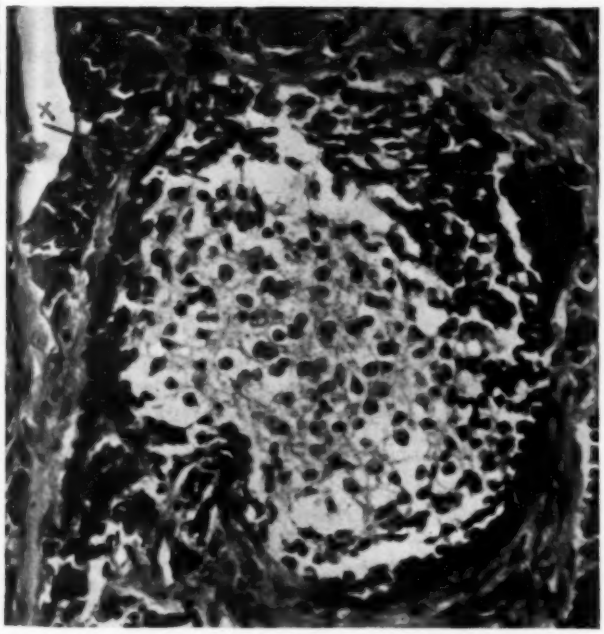

FIG. 752.—To show the differentiation of neuroblasts into glial cells. In the mid-zone cells resembling oligodendroglia are seen and at the upper pole of the glial differentiation unipolar spongioblasts are present at (X). H. and E. (× 170.)

(1932), Robinson (1937), and Thoma (1949) refer to and accept without question the case reported by Mummery and Pitts (1926) as a melanotic epithelial odontome in spite of the fact that it was a unique example necessitating a special subdivision of the group of tumour under consideration. We think that the melanotic gingival tumours of nervous origin should be sharply separated from epithelial odontomes.

The case of Mummery and Pitts is of similar age group—under six months—similarly pigmented, of comparable size, site, and histological picture. In their description, much attention is paid to the tooth caught in the centre of the tumour growth and primitive tooth elements were also noted, but such elements may be normal, being only displaced by the growing tumour at this child's age. They give a separate description of the soft part of the tumour, and every effort is made to identify this part with tumours of dental origin, whereas the account suggests

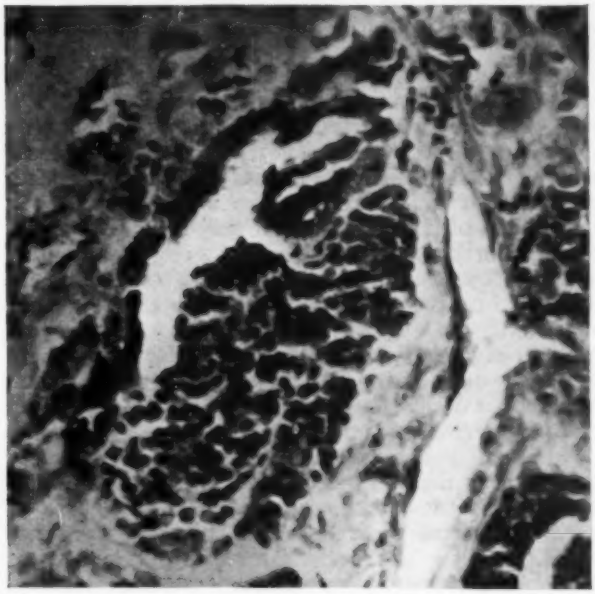

FIG. 753.—To show pigmented cells lining the clefts differentiating from the neuroblastic mass. H. and E. (× 170.)

that the soft melanotic part of the tumour contains elements of the same type as ours. Their Fig. 9 shows alveoli closely resembling nervous tissue and their Fig. 10 shows pigmented clefts lined by cubical epithelium. They describe their tumour as both a 'melanoma' and an 'epithelioma odontome'. Willis (1948, a) accepted it as essentially an odontome, but we are not convinced that the tooth elements are more than normal tissues, included within, and modified by, the tumour. We believe that the illustrations of Mummery and Pitts indicate that the essential elements are not odontogenic but of neuro-epithelial origin. The nature of this tissue and the source of the pigment demands some attention. In our case the pigment remained fast when the sections were treated with weak alkali or acid, hot or cold, and when attempts were made at bleaching with hydrogen peroxide and with hydrochloric acid on potassium chlorate in 70 per cent alcohol. It was bleached by 25 per cent potassium permanganate followed by 5 per cent oxalic acid and it was blackened by Fontana's diamino-silver carbonate. None of the pigment gave any reaction to iron. The pigment is considered beyond any reasonable doubt to be melanin.

Melanin is found in a variety of sites, including occasionally the buccal mucosa, from some of which melanomata are known to arise. As the tooth germs are derived from the dental lamina, itself an ingrowth of epithelial buds from the buccal mucosa, it might be supposed, theoretically, that a pigmented epithelial odontome or melanoadenomantoblastoma (Thoma,

1949) is not impossible; nevertheless, we consider that the existence of such a tumour is unproven and that the rare pigmented gingival tumour of infancy in all cases reported is of neuroectodermal origin and is derived from ectopic retinal elements.

Thoma (1949) reviewed the odontogenic and osteogenic tumours of the jaw and made as detailed a classification as that given by Churchill (1932), though not the same. Each reviewer recognized the very rare pigmented variety, and Thoma cited the case of Mummery and Pitts, but offered no critical comment. His photograph is a picture similar to our *Fig.* 749, and, we believe, is of similar nature.

Ectopic retinal tissue is known to occur in varying sites in the orbit (Verhoeff, 1922), but if the dislocation of primitive retinal elements occurs so soon after its early determination as to be moved caudally in the folding of the face, it is not unlikely that it might develop further in either the maxilla or the mandible, and thereby disturb the formation of the teeth, the resulting mass then presenting as a 'melanotic adamantinoma'. Jordan and Kindred (1942) show that the pigment of the future retina is present when the embryo is about 3 mm. in length in the proximal layer of the double-layered optic cup. It is probable that even before this the neural tissues have become determined, and if any displacement of these tissues has occurred the maturation process may nevertheless continue in these displaced tissues. The cause of such displacement is conjectural, but that such displacement does occur in other parts of the body is recognized and the theory is thus reasonable.

At this very early stage in embryonic development it is important to know if such displacement could reach the tissues eventually to be differentiated into the upper and lower jaws. This is possible at about the 2–5-mm. stage before the first branchial arch has begun to show a maxillary process. It is important that this arch should not yet have differentiated, as not only have five out of the six recorded tumours of this kind been found in one or other jaw, but two of them have actually been in the mandible, thus the displacement occurred before the maxillary process was formed or at about that time. The tumour in the case of Clarke and Parsons (1951), the sixth of the recorded tumours of this type, was situated in the anterior fontanelle of a nine-months-old male. It is clearly similar to ours in histology, and to compare it to the Cyclopian eye is irresistible. Anatomically it has been displaced upwards to grow in the region of the anterior fontanelle equally early in embryological development.

We therefore agree with Halpert and Patzer (1947), Clarke and Parsons (1951), and with Martin and Foote (1951), whose case of a four-month-old female may be claimed to be exactly similar to ours, that such tumours are of retinal origin. The histological evidence rests on comparison with the embryonic eye at an early stage—the 2·5-mm. fœtus onwards, where the retina is still composed of neuroblasts, and later when the optic cup has formed the similarity between it and our *Fig.* 753 is evident. The development of retina-like structures in ovarian and testicular teratomata is not very uncommon and is proof that heterotopic neuro-epithelial tissue can differentiate in this direction (Muir, 1952; Willis, 1948, b). Such findings support our belief that the pigmented gingival tumours of infancy are derived from neuro-epithelial rudiments differentiating towards retina and may perhaps indicate a fundamentally teratoid origin. Had the ectopic tissue continued to differentiate, an epignathus might have developed as these are essentially abnormalities of the skull base. Certainly a teratoma and an epignathus in this site have something in common in their origin. The histological picture rules out 'congenital epulis', though clinically there is much resemblance (Middleton and Harvey, 1933).

SUMMARY

A rare variety of congenital epulis is described and the literature pertaining to it is reviewed.

It is claimed that this tumour is derived from primitive neural ectoderm, and its development is discussed.

It is probable that some such tumours have hitherto been recorded as adamantinomata; grounds for doubting this diagnosis are brought forward.

We acknowledge with pleasure the help given to us and the interest shown in the case by Professor D. F. Cappell and by Dr. E. K. Dawson.

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THE RESULTS OF DENIS BROWNE'S OPERATION FOR HYPOSPADIAS

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The results of treating 121 cases of hypospadias by the 'buried strip' method described by Denis Browne (1949, a, b) are reported here, not only because they confirm that this method is more satisfactory than any other yet described, but in order to emphasize the importance of certain principles and technical details.

These 121 cases which have been operated on either by Denis Browne himself or by one of the registrars at the Hospital for Sick Children, Great Ormond Street, are a consecutive series and include all those cases of hypospadias requiring repair which have come under Mr. Browne's care since he started to use this method in 1946. Those cases of hypospadias which have had a very mild degree of deformity and have only required a meatotomy have not been included. The series contains several cases which had had unsuccessful operations elsewhere and one case which had had the prepuce excised. In *Table I* the results are set out according to the extent

Table I.—RESULTS

Degree of Hypospadias	*Perineal*	*Mid-scrotal*	*Peno-scrotal*	*Mid-shaft*	*Sub-coronal*	*Total*
No. of cases	11	16	30	46	18	121
No. with fistulæ	3	2	1	2	0	8

of the deformity and the number of cases in each group which developed fistulæ is recorded. These cases are classified according to the original position of the hypospadiac meatus and not the position after the initial operation to relieve the chordee, at which time the meatus was frequently slid back as much as half the length of the penis. The cases listed as perineal hypospadias were not hypospadiacs in the strict sense, but intersexes in whom it was necessary for one reason or another to form male external genitalia. All these cases had at least a rudimentary vagina, which either opened separately into the perineum or opened in a common orifice with the urethra. The term 'midscrotal' has been used for cases where the scrotum was cleft except for a small area posteriorly and the meatus situated well behind the base of the penis.

It will be seen from the Table that only 8 of these 121 cases developed a fistula post-operatively. In the other 113 cases the meatus was satisfactorily advanced to the end of the penis in one operation. In the earlier cases in this series no attempt was made to advance the meatus beyond the corona, but in the later cases it was advanced to the tip of the glans by denuding of skin two triangular areas on the ventrolateral surfaces of the glans and drawing flaps over these.

In considering the cases which developed fistulæ it is interesting to note the site at which they occurred. In 5 of the 8 cases the fistula was not in the midline where the flaps had been sutured but about ½ cm. lateral to this. They were at the site of one of the 'double stop' sutures. This was particularly well exemplified by one case which had two small fistulæ, one on each side of the midline exactly opposite to each other. There can be no doubt that the fistulæ in these 5 cases were caused by drawing a 'double stop' suture too tightly. When the post-operative swelling occurred the suture caused a small area of pressure necrosis followed by a small slough which left a fistula when it separated. These fistulæ should be regarded as avoidable and were due to errors in the operative technique. These cases emphasize the importance of using the 'double stop' sutures only as tension sutures and the fact that they must not be drawn tightly enough to oppose the skin edges of the flaps but only to hold them gently together when allowance is made for the post-operative swelling. They also show the importance of using the bead to spread the pressure without cutting off the blood-supply to the healing edges. Various other types of tension sutures were tried during the development of the method and no other was found to be satisfactory.

In the remaining 3 cases which developed a fistula, this was in the midline. The first case was one of perineal hypospadias where a small abscess developed around the posterior part of the new urethra. When this abscess discharged it left a fistulous connexion between the new urethra and the skin over the perineum. This abscess was probably caused by contamination from the anus at the time of operation, as a pure culture of *B. coli* was obtained. The second case was one where the perineal catheter slipped out of the bladder on the first post-operative day. An attempt was made to replace the catheter in the ward, but this was not successful. The child passed urine through the new urethra without difficulty and it was decided not to reinsert the catheter as a further anæsthetic would be required. It is now felt that this was a mistake. It is not easy to replace such a perineal catheter and yet it is important to do so. There is a tendency for the catheter to be passed out of the bladder even though it is carefully stitched to the perineal skin. If the catheter fails to drain and the bladder fills, it should be washed out. Examination may then show that the catheter is lying in the urethra and pointing towards the meatus. It must be removed and reinserted correctly. This tendency for a catheter to be passed out of the bladder is the reason that it is necessary to use a Malecot catheter. The third case was one of penoscrotal hypospadias which was straightforward until the 'double stop' sutures were removed on the eighth day. Almost immediately afterwards an ooze started in the urethra which developed into a considerable secondary hæmorrhage. This had the effect of filling the urethra with a large blood-clot which blew the suture line open for about 1 cm. The hæmorrhage stopped without further treatment, but the split remained as a fistula. In one other case a similar secondary hæmorrhage occurred on the eighth day, and in this instance

the hæmorrhage was controlled in theatre and the penis resutured. The urethra healed without a fistula.

The fistulæ in these 8 cases were closed in each instance with a single simple operation and all 121 cases are now passing urine satisfactorily from the end of the penis. At this operation a small incision is made just around the fistula itself through the skin, but not into the urethra. The skin is then dissected off the urethra and elevated for about 1 cm. in all directions. Traction is exerted on the fistula, pulling it out into a longer tube, and this is cut off flush with the urethra. This leaves a large defect in the urethra itself. No attempt is made to reconstruct the urethra as an internal tube, but the skin is sutured over the raw area using the ' double stop ' tension sutures and fine catgut to approximate the edges. In this way all the fistulæ have been closed and in no case has a stricture developed.

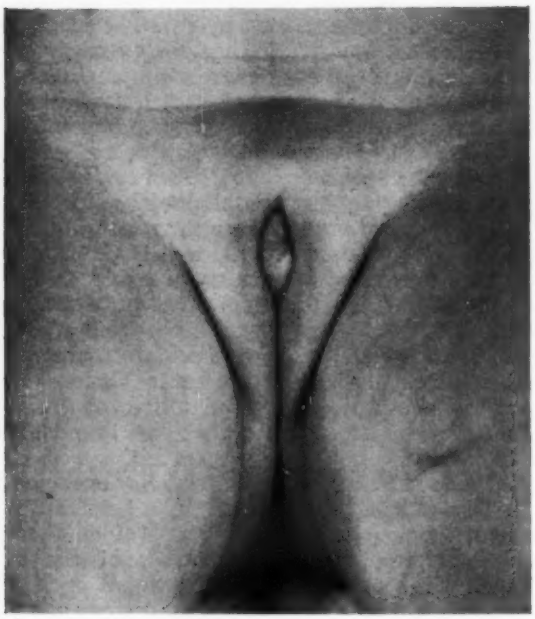

FIG. 754.—A case of perineal hypospadias (male intersex) before repair.

FIG. 755.—Same case after repair by buried strip method of Denis Browne.

There has been only one other complication of any real importance in this series and that was seen in two cases. In each of these cases the penis was very short. After the dorsal relieving incision had been made and allowed to expand laterally, it was seen that the incision had become much greater in a lateral direction than in length. This allowed the penis to heal with very little penile skin between the relatively inextensible abdominal skin and the corona, with a resultant tendency towards dorsal bowing. After the hypospadias was corrected and the swelling and œdema had settled, it was very simple to perform a V–Y plasty on the dorsal surface of the penis and relieve this deformity. It must be emphasized that this has only been seen twice in this series of 121 cases in spite of the fact that the dorsal relieving incision was made in every case from the base of the penis to the corona. In some cases it was made twice; first at the preliminary operation for straightening the chordee and again at the definite operation for advancing the meatus. The making of this incision is absolutely essential for the success of the operation for two reasons: First, it allows the preputial skin to migrate into the flaps. Thus this ideal reservoir of penile skin is used without any complicated operations such as Edmunds (1926) recommends. Secondly, it relieves tension on the suture line. Drawing the lateral flaps together over the buried strip on the ventral surface cannot be done without some tension, and this tension is greatly increased by the post-operative œdema. After the relieving incision is made the tension on the suture line can be seen to disappear. The post-operative œdema is lessened because the serum can escape from the raw surface and the flaps are sufficiently lax to allow erection to occur without detriment to the result. In no case in this series has any keloid developed on the dorsum of the penis as a result of this incision.

The only other difficulties encountered have been very slight. In one case the raw areas on the glans were made too close together, with the result that the meatus was too small. This was corrected by a meatotomy as an out-patient. Three cases developed a forked stream owing to a small bridge of skin dividing the meatus into two. This bridge was divided with a simple cut, again as an out-patient procedure.

In only 1 case has there been any trouble caused by erection. As already explained, erection does not cause undue tension on the suture line. However, in 1 case a severe erection occurred on the tenth post-operative day shortly after the catheter had been removed. This erection caused a retention of urine and considerable pain. Heavy sedation was given, the erection subsided, and urine was passed while the child was asleep. There was no further trouble and the patient was discharged well healed on the fourteenth post-operative day.

In no case in this series has the site of the perineal drainage persisted as a fistula. Usually a small amount of urine passes through this incision during micturition for two or three days after the catheter is removed. If this persists longer than two days a pad is pressed onto the perineum during the act of micturition. With this régime all these incisions have closed within seven days of the removal of the catheter.

FIG. 756.—Same case micturating after repair.

In cases of perineal hypospadias no attempt has been made to remove the vagina even when this has been well developed and capacious. It is drained separately with a small rubber catheter. The bladder drainage in these cases cannot be brought out through a separate incision, but the Malecot catheter is brought out through the suture line about 2 cm from the posterior end between two 'double stop' sutures. This has proved entirely satisfactory. *Fig.* 754 shows such a case before treatment and *Fig.* 755 after repair. *Fig.* 756 is the same child micturating. The penis is very short, but initially it was no more than an enlarged clitoris.

Examination of *Fig.* 757, which is a case of perineal hypospadias (proved to be a male intersex), shows that there is a strip of urethral mucosa running from the perineal meatus on to the under-surface of the phallus. This strip contains no hair follicles. If the embryology of the area is considered it will be realized that this strip is covered by the fusing of the inner genital folds during the development of the normal male urethra. It is, in fact, the normal roof of the urethra. The suggestion of Smith and Blackfield (1952) that this method of repair is unsuitable in cases of perineal hypospadias because it entails burying a strip of hair-bearing skin is entirely erroneous. Examination of such cases preoperatively, subsequent urethroscopy, and consideration of the embryology of the area all prove this criticism to be invalid.

In none of the 121 cases in this series has there been any stricture formation. The urethra is always supple and of wide bore. Sounds have been passed on a number of these cases when they have been seen at follow-up, and in each case it has been noted that the new urethra tends to be wider than the normal for a child of similar age and development. This observation has been confirmed in a few cases which have been urethroscoped at varying periods post-operatively. In each case the new urethra was seen to be a wide-bore tube with a smooth lining resembling penile skin rather than urethral mucosa.

FIG. 757.—Another case of perineal hypospadias (male intersex) showing the mucosa extending from the perineal meatus along the ventral surface of the phallus to its tip.

The site of the old meatus was identified by the change in the lining membrane, and this is usually the narrowest part of the urethra; a fact which emphasizes the importance of a preliminary meatotomy. The urethra has a circumference considerably greater than the width of the strip of skin which is buried. The experimental work of Nesbit, Butler, and Whittaker (1950), which was performed on the abdominal skin of animals, does not apply to the similar type of procedure performed using penile skin, as is shown by the experimental work of Johanson (1953) on the penis of dogs.

It is now seven years since the first cases were operated on by this method, and it can be seen that the new urethra grows with the penis without any tendency to cause a recurrence of chordee. The penis itself develops normally and appears to be functionally perfect, though there is no record yet of a case where it has been used for reproductive purposes.

In 1949 Johanson, visiting from Stockholm, examined the first 48 cases in this series and he has included his findings in his thesis on reconstruction of the strictured male urethra (Johanson, 1953). His report is particularly valuable as it is an independent survey of Denis Browne's results by a surgeon from another country.

SUMMARY

A consecutive series of 121 cases of hypospadias repaired by the 'buried strip' method of Denis Browne is reported.

Eight cases developed a fistula and all these were closed by a single operation.

In the other 113 cases the meatus was advanced to the end of the penis successfully in one operation.

The results are discussed.

I wish to thank Mr. Denis Browne for permission to study his cases, operate on some of them, and to publish these results; and to thank Mr. Derek Martin for his skill and care in providing the photographs.

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COEXISTENT SEMINOMA AND TERATOMA OF TESTIS

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THE coexistence in one testis of a seminoma and a teratoma as distinct and separate tumours is uncommon. Willis (1948) reported 5 cases and found 10 cases recorded in the literature. Of these 15 cases, 7 showed separate tumours, 6 showed some degree of mutual infiltration but clearly had been separate, and 2 showed extensive mutual infiltration. A further 20 cases are recorded in the literature. Ferrero (1921) described a testis in which there was a benign teratoma infiltrated to a small degree by an adjacent seminoma. An almost identical case was recorded by Bogliolo (1931). Mercier and Bourgue (1939) are cited by McDonald and Broders (1941) as reporting a specimen which contained three separate tumours—a teratoma, a seminoma, and a chorionepithelioma. Peyron (1939) mentioned 5 further cases but gave no details. Scully and Parham (1948) recorded a case in which there were two grossly distinct tumours. King (1952) reported 6 cases of discrete co-existent teratoma and seminoma, and 5 cases with some degree of mutual infiltration, but he gave details of only 2 of these cases.

Thus, in all, 35 cases have been recorded. In many of these reports little or no description is given of the specimens and in view of their rarity it is considered worth while to record a further case.

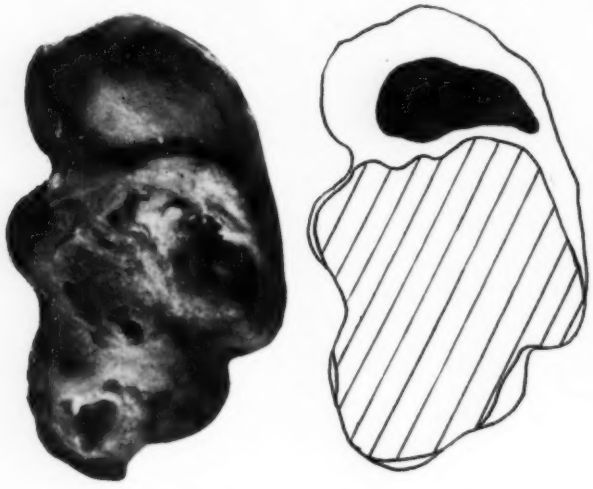

FIG. 758.—Vertical section of the testis with key diagram showing seminoma black and teratoma hatched. (× ⅓.)

CASE REPORT

The patient, a man aged 28, first noticed a swelling in the left testicle in April, 1951. It gradually increased in size, and was occasionally painful. An attack of pain in February, 1953, brought him to the Out-patient Department, when he was found to have a hard, craggy tumour involving the left testis and epididymis. The urine was normal, and excretion urography showed no abnormality of the renal tract. The Aschheim-Zondek test was positive. Left orchidectomy was performed by Mr. E. G. Muir on Feb. 19, and following operation a course of deep X-ray therapy was given.

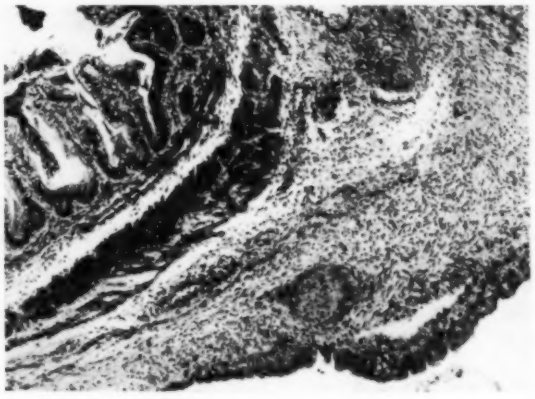

FIG. 759.—Teratoma showing intestinal epithelium with adjacent smooth muscle and respiratory epithelium with adjacent cartilage. H. and E. (× 46·6.)

THE SPECIMEN.—The specimen consisted of a testis together with the epididymis and part of the spermatic cord. The testis measured 4·2 × 2·8 × 2·5 cm. and showed several nodular protuberances on the external surface. Serial slices showed it to contain two distinct tumours (*Fig.* 758). The larger tumour, measuring 3 × 2·8 × 2·2 cm., was irregularly lobulated and occupied the lower two-thirds of the testis. Except where it extended into the epididymis for a short distance, it was surrounded by a thin rim of testicular tissue. Its cut surface showed areas of grey and brownish tissue with numerous small cysts. The smaller tumour, measuring 1·5 × 1·4 × 0·7 cm., was ovoid and situated in the upper pole. It was of a uniform white colour and entirely surrounded by testicular tissue, a narrow zone of which intervened between the two tumours.

Histology.—Although it appeared obvious macroscopically that the two tumours were distinct, many

blocks of tissue were examined from the two tumours and from the intervening zone of testis. The sections of the larger tumour showed it to be a malignant teratoma. It was composed mainly of well-differentiated adult tissue (*Fig.* 759), but there were also areas of less mature 'malignant' tissue located at the periphery of the tumour. Epithelium of stratified squamous, respiratory, and intestinal type was identified together with nondescript glandular structures and areas of poorly differentiated epithelial tissue which in places resembled an adenocarcinoma (*Fig.* 760). There were several areas of necrosis, and adjacent to some of them the poorly differentiated epithelial tissue showed swelling and fusion of cells, so giving an appearance often likened to that of a chorionepithelioma (*Fig.* 761). In addition there were various mesenchymal components including adipose tissue, connective tissue, cartilage, smooth muscle, nerve-bundles, lymphoid tissue, and undifferentiated embryonic mesenchyme. Growth extended into the epididymis for a short distance. Sections of the smaller tumour showed it to be a typical seminoma (*Fig.* 762). It was entirely separated from the teratoma by a narrow band of compressed testicular tissue (*Fig.* 763). The uninvolved testis showed tubular atrophy and prominent groups of interstitial cells. No growth was present in the upper cut end of the spermatic cord.

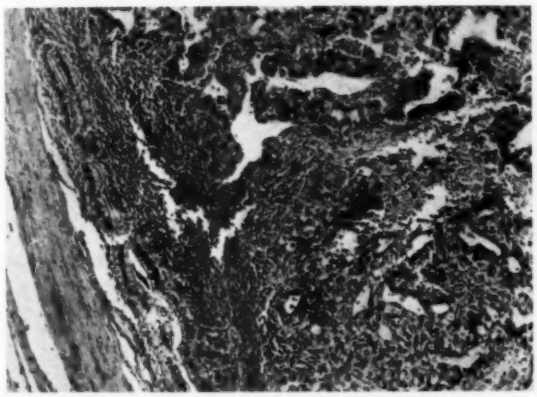

FIG. 760.—Teratoma showing glandular epithelium with adjacent poorly differentiated epithelial tissue. H. and E. (× 46·6.)

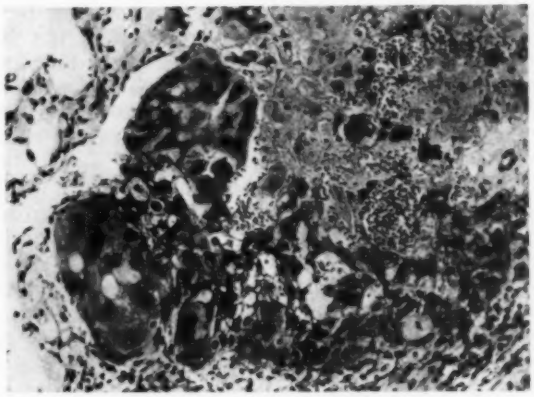

FIG. 761.—Teratoma showing area simulating a chorion-epithelioma. H. and E. (× 120.)

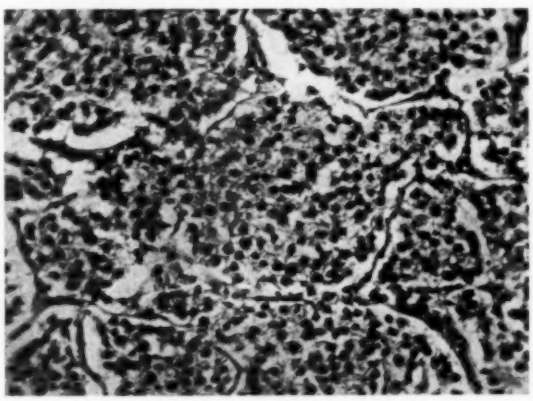

FIG. 762.—Representative area from seminoma. H. and E. (× 120.)

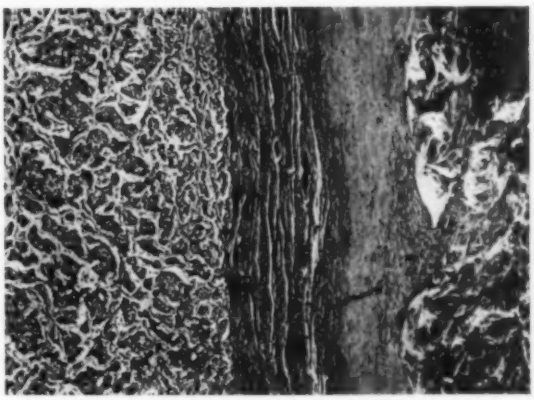

FIG. 763.—Narrow zone of compressed testicular tissue between seminoma (left) and teratoma (right). H. and E. (× 26·6.)

DISCUSSION

The most acceptable classification of testicular tumours currently available is that of Willis (1948.) Apart from interstitial-cell tumours and other rare tumours, two distinct types are recognized, the seminomas or seminal carcinomas, and the teratomas. Seminomas arise from spermatic cells of the tubules, have a uniform cellular structure, and are unrelated histogenetically to teratomas. The latter are of heterogeneous structure (similar to teratomas arising elsewhere in the body) and contain a variety of tissues not occurring in the normal testis. They usually contain areas of well-differentiated adult tissue, but many of them also contain areas of undifferentiated tissue and are malignant. Teratomas are truly embryonic tumours arising from foci of pluripotential embryonic tissues. They may be benign and composed of fully mature tissues or they may be malignant and contain all combinations of mature and immature tissue. From the standpoint of classification the separation and recognition of subtypes of these tumours is valueless and confusing—apart from a purely functional subdivision into benign and malignant types. As examples of the confusion that may arise, one may cite firstly the mistaking of undifferentiated epithelial tissue for, and identification with, seminoma, and secondly the identification of areas of hæmorrhagic undifferentiated epithelial tissue with chorionepithelioma.

Willis (1948) suggested that the coexistence of a seminoma and a teratoma is not uncommon, as he noted 5 cases in his series of 45 testicular teratomas and seminomas. His series, was, however, to some

extent selected, as also was that of King (1952), who found 11 examples in a series of 45 testicular tumours. As so few cases have been recorded by other observers it would seem that the association is uncommon.

In the present case there are two distinct and separate tumours within the testis, one a teratoma and the other a seminoma. The teratoma is clearly malignant, but the abundance of adult well-differentiated tissue in it suggests that it was originally benign and that malignant change has been fairly recent. Furthermore, the relative sizes of the two tumours strongly suggest that the teratoma arose first. The question whether the teratoma played any part in the causation of the seminoma cannot be definitely answered. However, in the recorded cases in which descriptions of the tumours are given it is clear that the teratoma precedes the seminoma. Peyron (1939) suggested that the teratoma induces a seminoma. Willis (1948) stressed the fact that a developmentally abnormal testis, such as an undescended one, is predisposed to the development of a seminoma, and he suggested that a testis containing a teratoma is also a developmentally abnormal one. A further interesting point is that in the present case and in several of the recorded cases the teratoma showed evidence of recent malignant change.

SUMMARY

The clinical and pathological features are recorded of a case in which the testis contained two separate tumours—a seminoma and a teratoma.

We wish to thank Mr. E. G. Muir for permission to publish the clinical records and both him and Professor H. A. Magnus for their interest and criticism. We are also indebted to Mr. W. Smith for the photographs.

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CONCOMITANT RUPTURE OF THE SPLEEN AND LEFT KIDNEY

BY D. H. TEASDALE, ROCHDALE

RUPTURE of the spleen and left kidney, without external wounding, is unusual and presents a different problem to the type of penetrating wound so often seen during the last war and chronicled by Gordon-Taylor (1952) in the BRITISH JOURNAL OF SURGERY Supplement on "War Injuries of the Chest and Abdomen". Closed simultaneous rupture of the spleen and kidney, being caused by considerable violence, is often accompanied by severe skeletal damage, and a high proportion of cases do not reach hospital alive. Neligan (1948), analysing cases of ruptured kidney, found that, between 1921 and 1940, 150,212 surgical cases were admitted to the London Hospital, of which 54 had a renal injury (0·03 per cent), and only 1 had an associated rupture of the spleen. Winsbury-White (1924) found 1 associated splenic injury in his series of 42 consecutive cases of renal injury. Guterbock, dealing with post-mortem material, found that out of 36 cases of rupture of the kidney, 13 had an associated rupture of the spleen (36·2 per cent), showing that the combination is seen in the more severe type of accident, and is frequently fatal. Marion, Mathieu, Perrin, and Francillon (1946), who recorded 4 cases of concomitant rupture of the left kidney and spleen, and one of the right kidney and spleen, stated that the mortality of cases described in the French literature reaching surgical care was 50 per cent.

CASE REPORTS

Case 1.—A draughtsman, aged 20 years, was admitted to St. James' Hospital at 4.15 p.m. on Dec. 3, 1949, having been knocked off his bicycle by a lorry. On admission he was conscious, the pulse-rate was 60 per minute, and the blood-pressure was 104/74. He was in considerable pain, and was very tender over the left lower ribs and in the left hypochondrium. He was given morphine gr. ¼ and A.T.S. 1500 units, a provisional diagnosis of fractured left lower ribs having been made.

Nine hours after admission the pulse-rate had risen to 96 per minute, he had vomited twice, and had passed a little blood-stained urine on attempting micturition. He was conscious, very pale, with cyanosed extremities, and the blood-pressure was too low to be accurately recorded. There was tenderness in all areas of the abdomen and in the left loin, and the left side of the abdomen was rigid. On percussion there was dullness in both flanks and on auscultation no bowel sounds were heard. A tentative diagnosis of ruptured spleen and either ruptured kidney or bladder was made, with possible associated intestinal injury. He was transfused rapidly with two pints of Group O blood and laparotomy was performed an hour later.

AT OPERATION.—Under general anæsthesia, in the operating theatre, catheterization was performed, and 15 oz. of heavily blood-stained urine were withdrawn. A left para-umbilical paramedian incision was made, revealing about two pints of blood and clot in the peritoneal cavity. This was evacuated and the blood was seen to be coming from a diffusely lacerated spleen (*Fig.* 764), which was removed without difficulty, once the incision had been prolonged upwards. Careful search throughout the abdomen showed no further sign of injury apart from a retroperitoneal hæmatoma around the left kidney. The right kidney felt normal. The left kidney outline could be palpated through the hæmatoma and it appeared to be intact. During the course of the operation this hæmatoma did not increase in size, and it was, therefore, decided not to perform a nephrectomy and the abdomen was closed in layers. Four pints of

blood were transfused while the operation was in progress, and once the splenic pedicle had been secured the patient's general condition improved considerably, the blood-pressure at the end of the operation being 110/60.

POST-OPERATIVE COURSE.—Twelve hours later the general condition was good, the blood-pressure being 110/80. As no urine had been passed 20 hours after operation, catheterization was performed and 33 oz. of dark, stale, blood-stained urine were withdrawn, and the bladder was washed out with saline. Radiological examination showed fractures of the left 8th and 9th ribs, but no other bony injuries. Daily catheterization was required as he was unable to pass urine naturally. A neurological examination on the fourth post-operative day showed bilateral extensor plantar responses, an absent bulbocavernosus reflex, and brisk ankle-jerks. A diagnosis of concussion of the conus medullaris and sacral nerve-roots was made, with resultant neurogenic bladder.

FIG. 764.—The damaged spleen, showing two wide lacerations.

Lumbar puncture produced a clear fluid at a pressure of 120 mm. of water, and which was normal on laboratory examination.

In view of the neurological signs, cystoscopy was performed on the seventh post-operative day and it revealed an open bladder neck, the verumontanum being well seen as the cystoscope was withdrawn. As a neurogenic bladder was confirmed, a suprapubic cystostomy was performed at once. He was seen by the consulting neurologist, Dr. Redvers Ironside, on Dec. 22, who reported: "This patient appears to have sustained damage (contusion) to his spinal cord as evidenced by the bilateral extensor plantar responses. He has sensory changes over the distribution of the sacral 4th and 5th roots. He is likely to continue to show signs of recovery, but may be left with a tendency to dribbling, although the 3rd sacral root appears to have escaped serious damage."

He began to pass urine naturally on Jan. 6, 1950, and the suprapubic tube was removed on Jan. 8. An intravenous pyelogram outlined a normal right kidney and on the left the lower calices were shown, but in the region of the upper pole part of the dye appeared to have diffused out into the tissues (*Fig.* 765).

The suprapubic wound was dry by Jan. 20 and he was discharged home on Jan. 28.

FOLLOW-UP.—Subsequent pyelograms have shown a left kidney which is diminished in size and yet appears to function well (*Fig.* 766). When last seen on April 17, 1952, two and a half years after the accident, he was well, there was no dribbling, the blood-pressure was 130/70, and there were no abnormal neurological signs.

Case 2.—A male, aged 23 years, was admitted to St. James' Hospital on Sept. 11, 1952, at 10 a.m., having fallen 60 ft. from the roof of a bus garage. He was conscious, restless, pale, and sweating. He had bruising of the scalp, a compound fracture of the left elbow, and there was marked tenderness in the left loin, the left hypochondrium, and on springing the pelvis. The blood-pressure was 135/80. He was given 1 pint of Dextran, and 2 pints of blood by rapid transfusion, and morphine ¼ gr. Despite this his general condition deteriorated and he became extremely restless. The blood-pressure fell to 80/50 and he died two hours after admission.

POST-MORTEM EXAMINATION (Dr. C. Keith Simpson).—"External marks of injury were confined to bruising of the scalp over the left ear, and a compound fracture of the left elbow, with the tip of the ulna protruding through a split in the skin. The skull showed no fractures. There was blood in the bronchial tree, the result of a contusion of the left lung. The peritoneal cavity showed considerable fresh bleeding from a rupture of the spleen. The liver was normal. The right kidney was normal, but the left was split through the hilar region. There was a fracture of the left superior ramus of the pelvis with separation at the symphysis."

COMMENT

Aetiology.—There are a number of ways in which an injury of this character may be produced, but the common factor to all of them is a prodding force of considerable severity, applied over the splenic and left renal area. The lesion in both organs is a diffuse splitting from hilum to periphery. At the present time the commonest cause for this accident is direct impact with part of a moving vehicle, although a number of cases are still caused by falls on to protruding objects. In the series of Marion and others (1946), 1 patient fell on to his left flank from a height, 1 fell from his bicycle on to a steel stake, 1 was sandwiched between a lorry and a tram, and the last was hit by a car.

Diagnosis.—A ruptured kidney is usually suspected, since 85 per cent of the cases have hæmaturia, and there is a danger that the associated rupture of the spleen may be missed. When a ruptured left kidney has been diagnosed, several features may arouse suspicion as to the integrity of the spleen:—

1. A degree of shock out of proportion to that usually found in simple rupture of the kidney.

2. Signs of peritoneal irritation, the most important being tenderness in the abdomen, particularly rebound tenderness, and abdominal rigidity.

3. The presence of one or more fractured left lower ribs.

Renal rupture alone very rarely produces intraperitoneal bleeding over the age of 10 years old, the perinephric fat being an adequate protective in the adult.

Treatment.—If suspicion exists that there is associated intraperitoneal bleeding, it is wise to perform a laparotomy without undue delay. Ruptured spleen is one of the few conditions left in surgical practice where it is not justifiable to wait for the patient to be resuscitated before operation since the patient will often lose blood as fast as, or faster than, it can be replaced. *Case* 2 died before he could be operated upon, and it is in this type of case that intra-arterial transfusion might have been of value.

In *Case* 1 the main problem, the spleen having been removed, concerned the management of the ruptured kidney. It was not exposed because there was no palpable gross laceration and the bleeding appeared to have ceased. The risks of leaving the kidney were; recurrence of the hæmorrhage, sepsis, late hydronephrosis, and atrophy, possibly associated with hypertension. In fact none of these occurred, although the pyelogram subsequently showed more damage than was thought to be present at the time of operation. The majority of cases of ruptured kidney, in the absence of other injuries, have been managed conservatively for many years, with good results. Sepsis in the hæmatoma can now be averted by the use of antibiotics, a further reason for the avoidance of hasty nephrectomy. If a laparotomy is performed for an associated abdominal injury, when this has been dealt with, the temptation to remove a damaged kidney is considerable. Obviously, if the organ is fragmented, or if the overlying peritoneum is torn, or if the perirenal hæmatoma is enlarging, then nephrectomy is the only safe course, but if the damage is less severe, removal means the sacrifice of a valuable organ and increases the operative mortality. In the series of Marion and others (1946) nephrectomy was performed in every case, together with the splenectomy, with a 25 per cent mortality.

The other interesting feature in *Case* 1 was the associated contusion of the spinal cord and sacral nerve-roots, from which the patient made a complete recovery.

FIG. 765.—Intravenous pyelogram one month after injury, showing damage to the upper half of the left kidney, evidenced by irregular extravasation of dye, only the lower calix being distinguishable.

FIG. 766.—Intravenous pyelogram ten months after injury, showing a left kidney, diminished in size but functioning well.

I should like to thank Mr. H. K. Vernon and Mr. N. C. Tanner for allowing me to publish these cases, and for their advice and interest. I am also grateful to Dr. C. Keith Simpson and Dr. Redvers Ironside for their reports; Dr. R. L. Quilliam for the radiographs; and Miss E. Mason for the photography.

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TANTALUM REPAIR OF VERY LARGE INCISIONAL HERNIA

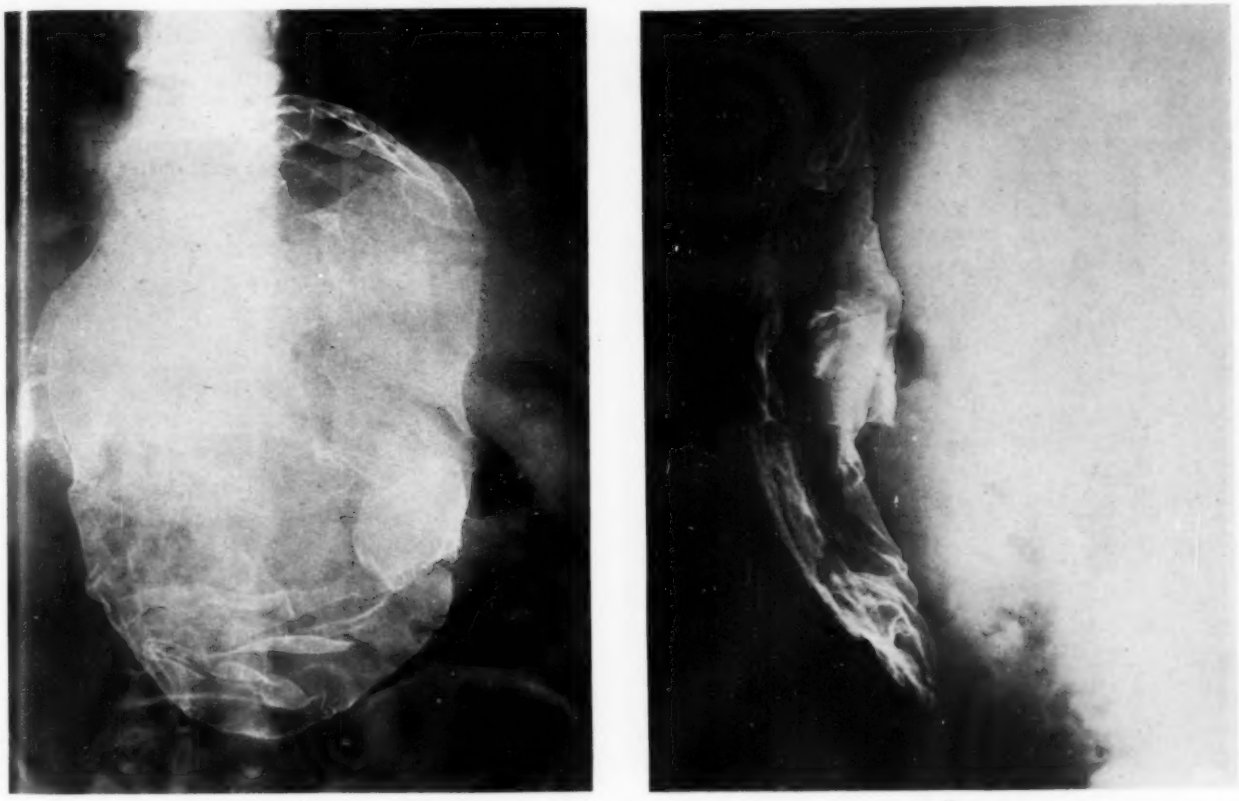
HEALING AFTER SECONDARY REPAIR

By A. J. COKKINIS and A. F. BROMWICH, Wembley

The repair of a very large incisional hernia can be a formidable undertaking. Cases under consideration are those in which the defect is so great that it is impossible to close it with muscle or other local tissues, and where the use of cutis grafts or fascial darns is either technically impossible or likely to end in failure. The enormous gaps occasionally resulting from breakdown of laparotomy wounds are comparable to congenital absence of the abdominal parieties, or to defects produced by resection of tumours of the abdominal wall.

In corpulent patients whose flabby abdomens are specially vulnerable, such a hernia cannot be controlled by belt or corset, and total disablement is to be expected unless the gap can be repaired by operation. It is obvious that some form of foreign material must be used. *Tantalum* gauze has certain important advantages (Lam, Szilgayi, and Puppendahl, 1948): It is strong, pliable, and inert, and can be used in the presence of infection (Koontz, 1951). Similar advantages are claimed for Fortisan fabric, an inert brand of regenerated cellulose (Nerat and Khedroo, 1952), but this appears to have been used only experimentally.

A number of technical points must be observed when a tantalum insertion is made. The cutis and subcutis should be separated from the fascia for at least 5 in. beyond the margins of the defect; the actual edges are more easily determined by palpating from within the peritoneal cavity. The defect is enlarged until firm muscular or aponeurotic structures are reached. A bed is made for the marginal 1½–2 in. of the tantalum (a double piece being used when possible) by dissecting apart the two layers into which the abdominal wall naturally falls. The edge of the tantalum is fixed securely by non-absorbable and inert sutures to the firm aponeurotic rim at the edges of the dissection. The exposed part of the tantalum is covered by a single fascial graft or fascial strips, the redundant part of the skin is excised, and the skin edges are brought together without tension. The wide exposure of tissue, the unhealthy condition of the skin, and the insertion of foreign material may combine to defeat the strictest aseptic technique and the best antibiotic cover. A small local breakdown of the wound is a likely result, and the battle against infection which follows is often a long and unsuccessful one.



A B

Fig. 767.—Radiographs of tantalum gauze in situ. A, Anteroposterior view. B, Lateral view.

It is a generally accepted surgical principle that an infected wound containing a foreign body will not heal until the foreign body is removed. With the advent of such inert materials as tantalum this principle requires modification. The following case indicates, firstly, how large a defect can be repaired with tantalum gauze, and, secondly, how the non-reactive properties of tantalum enabled deliberate closure of a large skin defect with infection to be carried out without removing the tantalum gauze.

CASE REPORT

W. H. L., an obese male aged 65, was admitted to hospital on Oct. 6, 1951 following a road accident. Laparotomy was performed the same day, rupture of the spleen being suspected clinically. A long left paramedian incision was made, but no intraperitoneal lesion was found. Post-operative chest complications (several ribs were fractured) led to disruption of the wound on the sixth post-operative day. Secondary suture was carried out and further convalescence was uneventful. He reported again in January, 1952, with a very large incisional hernia. A wide gap could be felt from the costal margin to 3 in. below the umbilicus. The hernia was not controllable with belts or corsets and a repair operation was undertaken on April 3.

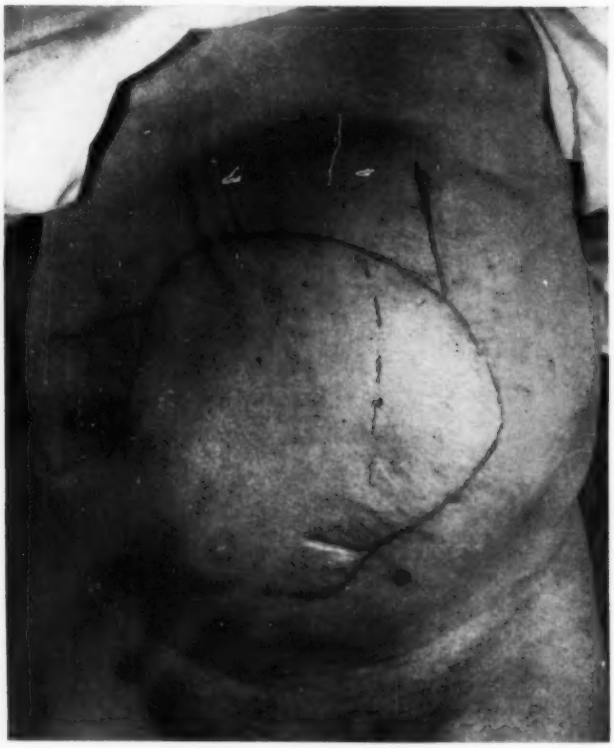


FIG. 768.—Abdomen of patient with scar marked out. Dotted lines indicate approximately original site of flap. Vertical scar on left shows upper portion of original incision for repair of hernia.

AT OPERATION (A. J. C.).—All scar tissue was excised and the peritoneal layer repaired with overlap. A defect of all layers of the abdominal wall measuring 9 in. by 5 in. was left. The margins of the defect were split by dissection and two pieces of tantalum gauze, each 12 in. by 6 in., were inserted one above the other with overlap and fixed to the aponeurotic bed with a large number of nylon sutures (*Fig.* 767). The thin and atrophied rectus sheath was drawn over the gauze and the remaining gauze was covered with a free graft of fascia lata (Lam and others, 1948). The skin was pared and the incision closed with tension.

A low-grade deep wound infection became apparent three weeks after operation, and a small part of the wound broke down. An attempt to resuture the wound on July 3 failed, and the patient was left with a skin defect about 4 in. by 3 in., the floor of which consisted of tantalum gauze embedded in, but not covered by, granulation tissue. Infection was, of course, present, the organisms varying with each change of antibiotic and of local dressing. Further operation was undertaken for the repair of this defect on Aug. 21.

SECOND OPERATION (A. F. B.).—The scar tissue surrounding the defect was excised and a large skin-flap about 8 in. in diameter was cut from the abdominal wall to the right of the defect and based inferiorly. This was rotated through about 30° over the defect and sutured in place (*Fig.* 768). The secondary defect, which was well clear of the tantalum, was closed by approximation and a small drain inserted through a stab wound to obviate hæmatoma formation. A minor breakdown of the closure of the secondary defect was remedied by a split skin-graft, and thereafter convalescence was uninterrupted.

The patient was last seen in October, 1953, when his abdomen was soundly healed and he had returned to work.

COMMENT

A defect of the abdominal wall of the size present in this patient is virtually impossible to repair by any method other than insertion of tantalum gauze or similar material. There is no possibility of closure by using local tissue, and repair by fascial darn would be extremely difficult technically and result in a much less secure repair.

When the wound broke down after the primary operation the question of removing the tantalum was raised. This would have been impossible as the patient would then be left with a large defect of all layers of his abdomen, including skin—a condition not compatible with life. The decision to attempt secondary repair gains support from the experience of Koontz (1951), who describes cases of tantalum repair with wound infection which nevertheless healed with little trouble, and also, from another case seen elsewhere, where a persistent sinus following a tantalum repair of an inguinal hernia was shown to be due to a silk ligature and healed immediately after its removal.

CONCLUSION

It is again shown that large tissue defects need not deter the surgeon from operating for the relief of large ventral herniæ.

It is also shown that tantalum does not act as a foreign body even in the presence of infection.

SUMMARY

A case of ventral hernia repaired with a large sheet of tantalum gauze is described. Wound breakdown after this operation was repaired with a rotation flap, resulting in complete cure. The implications of this on a surgical principle are discussed.

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FRACTURE OF THE BRONCHUS

By T. A. SALE, Wirral

Fracture of the bronchus is an uncommon sequel to crush injuries of the chest. It may occur more commonly than is at present realized. A substantial number of reports have now appeared showing that the condition is not necessarily fatal. This report is of two such cases. Both recovered and further successful treatment was carried out later.

CASE REPORTS

Case 1.—The patient was a female aged 3. She was admitted to hospital on Jan. 13, 1950. She had been found lying face downwards under the wheel of a heavily laden waggon. She was " black in the face " and her mouth was full of blood. Examination on admission showed that she was deeply cyanosed, with marked dyspnœa. She expectorated small quantities of blood from time to time. There was bruising of the right chest anteriorly and posteriorly. Clinically there was a right-sided pneumothorax. The fauces had no bleeding point. Radiography of the chest showed a right pneumothorax (*Fig.* 769). The lung was not completely collapsed. No ribs were fractured. There was no surgical emphysema.

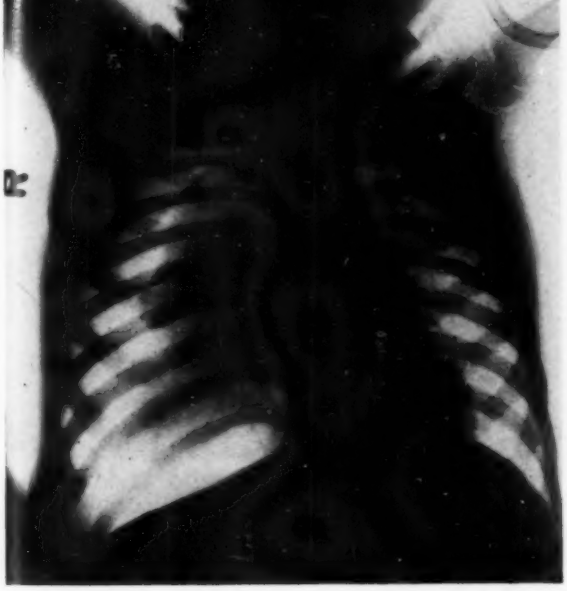


Fig. 769.—*Case* 1. Films taken on admission, showing right-sided pneumothorax, partially collapsed lung, and mediastinal displacement.

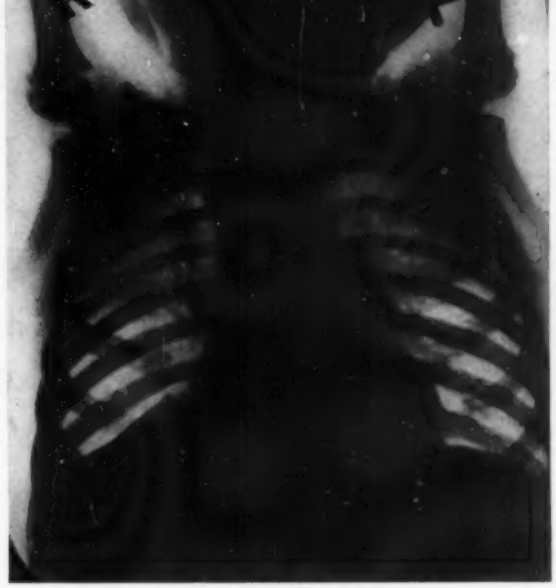


Fig. 770.—*Case* 1. Chest three months after injury, showing collapsed right lower lobe.

She was put in an oxygen tent, and given penicillin 125,000 units every four hours. She gradually improved and oxygen was no longer necessary after five days (Jan. 18). Serial films of the chest showed that the lung had gradually expanded, but the right lower lobe remained collapsed (*Fig.* 770). Further investigations were negative.

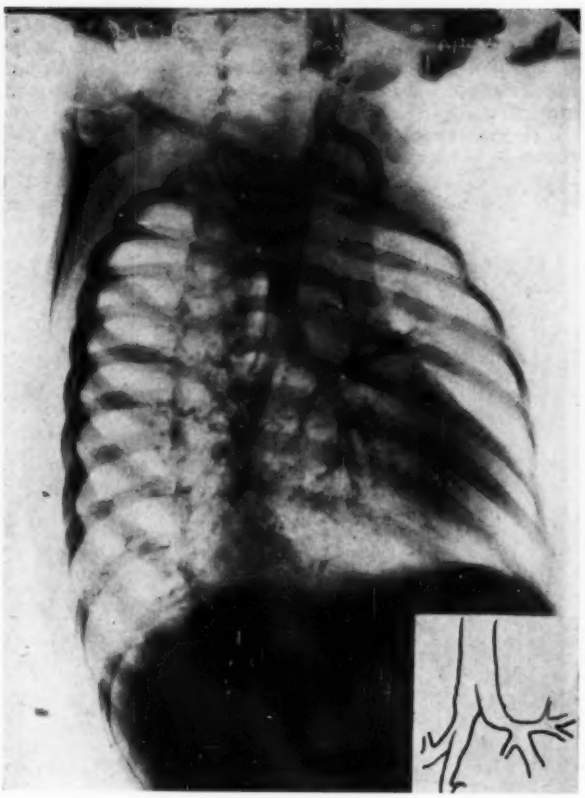
On Feb. 15 bronchoscopy was performed by Mr. F. Ronald Edwards. This revealed œdema and narrowing of the right main-stem bronchus. There was a piece of partially organized blood-clot in the lumen, which was completely occluding the right middle and lower lobe orifices. The clot was removed and sent for section. After removal of the clot a clear view of the right middle- and lower-lobe bronchi could not be obtained owing to the œdema. This confirmed the diagnosis of fracture of the lower part of the right main-stem bronchus. Section of the clot showed friable infected blood-clot with some organization. Postural drainage was instituted without any effect.

Bronchography was later performed (*Fig.* 771). It showed blockage of the right lower bronchus with the typical ' rat-tail ' appearance. As the changes in the right lower lobe were considered to be permanent, thoracotomy was advised. She was admitted to the Liverpool Chest Surgical Centre in June, 1950. On June 27 thoracotomy was performed. The chest was opened posterolaterally through the bed of the sixth rib. A few adhesions were present between the upper and lower lobe. The right middle and lower lobes were found to be collapsed, very small in size, and grey in colour. There was much fibrosis in the hilar structures of these two lobes. It was thought that a dissection lobectomy would be very hazardous and it was decided that a tourniquet should be applied. After a certain amount of dissection was carried out and the pedicle of the two lobes was adequately cleared, a high tourniquet was applied and lobectomy performed. Convalescence was uneventful.

The resected specimen showed typical collapsed lobes. The bronchi were also collapsed, but were otherwise normal. Microscopical sections of both lobes showed simple absorption collapse but there were no bronchial abnormalities.

Case 2.—This patient was a male aged 7. He was admitted to hospital on June 22, 1941, after being injured by an iron bar falling on to his chest and pinning him to the ground. When admitted, he had marked surgical emphysema from the face to the groin. He was markedly dyspnœic. He was treated by tracheotomy and the immediate effect was a marked improvement in his respiration. The chest showed on radiological examination a left-sided pneumothorax. His general condition improved and he was discharged from hospital on July 8. Observation showed that the left lung remained completely atelectatic and on May 24, 1946, he was admitted to the Liverpool Chest Surgical Centre. He was coughing up about 2 oz. of yellow non-offensive sputum every day. There was a little clubbing of the fingers. Clinically and radiologically he had an atelectatic left lung (*Fig.* 772). Bronchoscopy showed that the laryngeal opening was drawn over to the left. The left main bronchial opening was not seen, but was evidently some tiny opening to the left of a widely dilated right main bronchus. Lipiodol was not instilled as the right bronchial tree was so moist. A left-sided bronchogram showed bonchiectasis of the left lung. On Aug. 1, 1946, thoracotomy was performed. A few adhesions were present. The lung was easily freed. There were many slightly enlarged glands around the hilum and these obstructed a dissection pneumonectomy. A tourniquet resection of the left lung was therefore performed.

Post-operatively he developed infection in the left pleural space, which cleared with aspirations, drainage, etc. Examination of the resected lung showed a shrunken lung with old pleurisy on the surface. There were adhesions between the lobes. There were many glands at the hilum. There was gross tubular dilatation of the bronchi of both lobes. Histologically, there was gross peribronchial fibrosis and infiltrations with chronic inflammatory cells. The lumina of the bronchioles were filled with pus. The pleura showed a fibrous thickening. Follow-up has been most satisfactory.



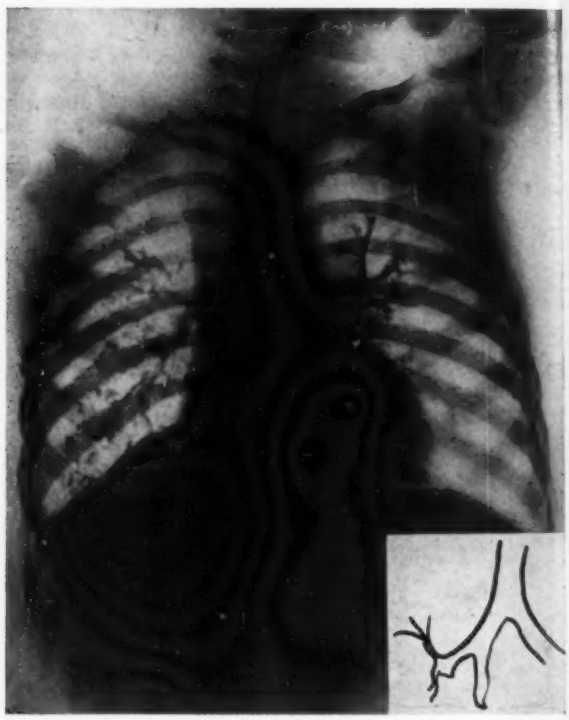


FIG. 771.—*Case* 1. Bronchograms showing 'rat-tail' bronchus.

DISCUSSION

Krinitski (1927) was the first to report a case of spontaneous recovery from this type of lesion. Of 38 cases collected by Kinsella and Johnsrund (1947) 19 had recovered spontaneously. Six further cases have been reported. The rarity of the condition and the unusual features of these two cases have prompted this presentation.

The condition is found at all ages, but mainly in young men and children. The exact mode of fracture is still undecided. It is well established that the laceration in the bronchus is not due to direct trauma by bone ends. Four theories are put forward. Thus it is said to be due to a simple shearing process acting on the lung. It is suggested that it may be the result of compression of a bronchus against the vertebral column. Schonberg's theory (1912) is that with the glottis closed the bronchus is rendered more rigid and therefore more likely to rupture when the chest is compressed. Kirkpatrick (1950) believes that when the chest is crushed with the glottis, the two air-filled lungs are pushed apart by the approach of the sternum to the spine.

The site of fracture is usually described as occurring at or near the carina. Kinsella and Johnsrund described a case with the usual injury in association with a second tear in an upper lobe bronchus. Fawcett's (1949) case had the lesion in the left lower lobe bronchus. The first case reported here is an exception to the general finding.

In most of the early case reports the condition was not suspected for some time after the accident.

Earlier diagnosis will become more common if the lesion of a fractured bronchus be kept in mind when dealing with compression injuries of the chest, particularly where mediastinal emphysema is manifest. The diagnosis is readily confirmed by bronchoscopy, and in the late case by lipiodol bronchography.

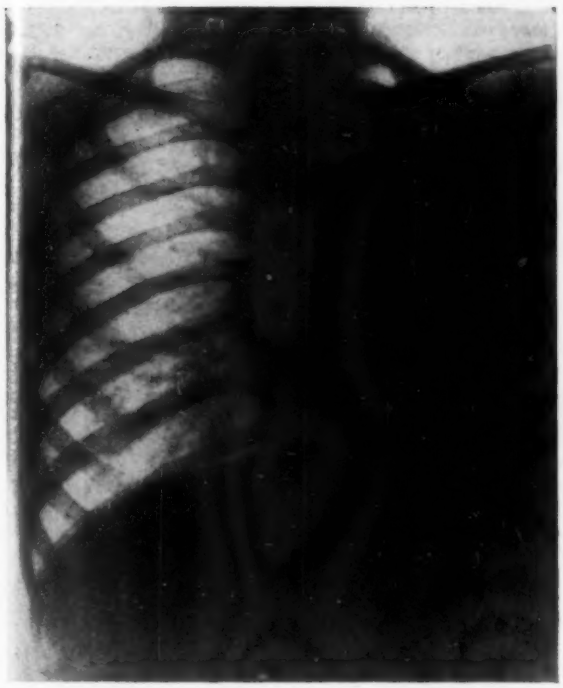


FIG. 772.—*Case* 2. Radiograph of patient showing atelectasis of the left lung.

Early thoracotomy, when necessary, will solve all doubts (Kirkpatrick, 1950; Scannell, 1951).

The treatment is divided into two main phases: The first, or acute, phase is due to the effect of the pneumothorax on respiration, and it is this which demands attention. The usual resuscitative measures of anti-shock therapy and oxygen administration combined with early antibiotic medication are taken. In many cases the patient will improve, as in the first case reported here. If a tension pneumothorax develops, aspiration or drainage may tide the patient over this phase (Jones and Vinson, 1939). If this fails, Sanger's (1945) dictum is that an immediate thoracotomy should be performed, and both Kirkpatrick and Scannell have opened the chest shortly after the injury was sustained and dealt with the lesion with most satisfactory results. It is the opinion of most authors that an increase in surgical emphysema aggravating the effects of the pneumothorax requires suprasternal incision. Carter and Guiseffi (1951) have used tracheotomy with success in other crush injuries of the chest. As shown in the second case report, tracheotomy can be used in fracture of the bronchus, as it had a most dramatic effect in easing respiratory embarrassment and was in fact a life-saving procedure.

When the patient is over the acute phase bronchoscopy and bronchography should be done to assess the problem remaining. Bronchoscopy, if done early enough, may help to re-aerate the lung by removing the occluding blood-clot (Rubin and Rubin, 1951). Resection of the lung appears to be the usual outcome of such accidents, as in the two cases reported here. Paulson (1951), however, after quoting the experiments of Tannenberg and Pinnier (1942), has made the suggestion that some of these lungs can be 'salvaged' by the use of various bronchoplastic procedures. He has had one such success and postulates that repairs such as that of Gebauer (1950) and Belsey (1946) may be tried. The experimental work of Taffel (1940) and Bucher, Burnett, and Rosemond (1951) may help many other such reconstructive operations. The difficulty of such operations must not be underestimated. Because of the adhesions and hilar glandular enlargement present in the cases here, resection could not be performed by the usual dissection methods but recourse had to be made to tourniquet resection. The usual residual deformity is a bronchial stenosis. Dilatation of such a stricture has had partial success (Piaget, 1939; Loffler and Nager, 1941). It appears that excision and primary suture of these obstructions is the method of choice, as well shown by Griffith (1949), if there is no permanent damage to the distal lung.

I am most grateful to Mr. F. Ronald Edwards for his help in the preparation of this paper and for his kindness in allowing me to present the case reports.

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A CASE OF THE BUDD-CHIARI SYNDROME: A CLINICO-PATHOLOGICAL STUDY

By E. G. JONAS and I. H. LAWRENCE

ORIGINALLY described by Chiari in 1899 as an 'endophlebitis obliterans hepatica', subsequent workers (Nishikawa, 1918; Berk, 1932; Rigdon, 1933) have shown that the thrombotic process in the hepatic veins is often associated with a similar process in the inferior vena cava. Perusal of the literature (Armstrong and Carnes, 1944; Hirsh and Manchester, 1946; Thompson, 1947; Brontë-Stewart and Goetz, 1952) gives the impression that this latter observation has gradually insinuated itself, and has been widely accepted into the definition of the Budd-Chiari syndrome.

This presents no difficulty, since the crux of the problem lies in the effects that are produced in the liver and portal circulation. Caval obstruction constitutes a complication which, though formidable, is in itself far less of a danger to life (Bennett, 1950).

As hitherto the Budd-Chiari syndrome has been a condition primarily of pathological interest, our observations on a further example of this condition have led us to suggest a more clinical approach to it.

CASE REPORT

HISTORY.—The patient was a single girl aged 19 years. In March, 1952, she was admitted to hospital after two hæmatemeses of bright-red blood. Her general health prior to this episode had been good, and there was no history of dyspepsia. About one year previously she had had a small hæmatemesis, which had been treated at home. It was at about this time that she first noticed prominent subcutaneous veins in the flanks. Other points worthy of note in her previous medical history were: (1) a congenital dislocation of the right hip; (2) a chronic skin eruption since the age of 6, affecting most parts of the body; (3) meningitis at the age of 10; and (4) mild bronchial asthma since the age of 12.

The family-history was not significant, but the patient had a twin brother, 6 ft. tall, who had always enjoyed good health.

FIG. 773.—Photograph of the patient's trunk, showing the superficial venous plexuses in the flanks. The scar of the incision for exposing the right sapheno-femoral junction (venogram of inferior vena cava) is shown.

She had responded well to blood transfusion and iron therapy, but was admitted to the Manchester Royal Infirmary, under the care of Dr. J. F. Wilkinson, on Aug. 2, 1952, for further investigation as to the cause of her hæmatemeses.

ON EXAMINATION.—On admission the patient was rather thin and underdeveloped, with a general increase in skin pigmentation, especially around the trunk. There was a mild clinical anæmia, but no jaundice. She was afebrile.

On the face, shoulders, arms, and lower limbs there were many scaly, papular skin lesions, most of which were discrete, though a few on the buttocks and thighs were confluent.

Chest expansion was symmetrical, and the trachea was central. The percussion note, breath-sounds, and vocal resonance were impaired at both lung bases. There were medium- and high-pitched rhonchi in all areas. No cardiac abnormality was found. Blood-pressure was 110/80.

The abdomen was protuberant, but no signs of free fluid were present. On both anterolateral aspects of the abdominal wall there were very large, tortuous superficial veins extending from the iliac crests to the breasts (*Fig.* 773). Inspection of the back showed a network

of similar veins between the buttocks and scapular angles. The flow in all the veins was cephalad. There was no caput medusæ, and indeed, the anterior abdominal wall was conspicuously free from dilated veins.

The liver was enlarged 7 cm. below the right costal margin, and was smooth, firm, and non-tender. The spleen was enlarged 11 cm. below the left costal margin.

Proctoscopy disclosed the presence of moderately large internal hæmorrhoids.

She had a right-sided optic atrophy and a divergent squint, due to right medial rectus muscle paresis.

The only other clinical abnormalities were a moderate dorsolumbar kyphosis, some limitation of flexion and of abduction of the right hip, and a true shortening of the right leg of 1 in.

INVESTIGATIONS.—Blood-count on admission—red cells 4·5 millions per c.mm., hæmoglobin 11·8 g. per cent (80 per cent Haldane), colour index 0·88, white cells 6800 per c.mm., with 11 per cent eosinophils. Subsequent counts showed no significant change, the eosinophils ranging from 4 to 13 per cent.

Urine: no abnormality. Blood-urea: 28 mg. per cent.

Liver-function tests: March, 1952—serum-bilirubin 0·1 mg. per cent, serum alkaline phosphatase 9·5 units per cent (King-Armstrong), thymol turbidity 0 units. August, 1952—serum-bilirubin 0·9 mg. per cent, serum alkaline phosphatase 10 units per cent, serum-albumin 3·2 g. per cent, serum-globulin 2·8 g. per cent. October, 1952—Bromsulphthalein test showed dye retention of 1·3 mg. per 100 ml. in the 45-minutes specimen, after injection of 5 mg. bromsulphthalein per kilo body-weight (normally, there should be no retention of dye in the 45-minutes specimen).

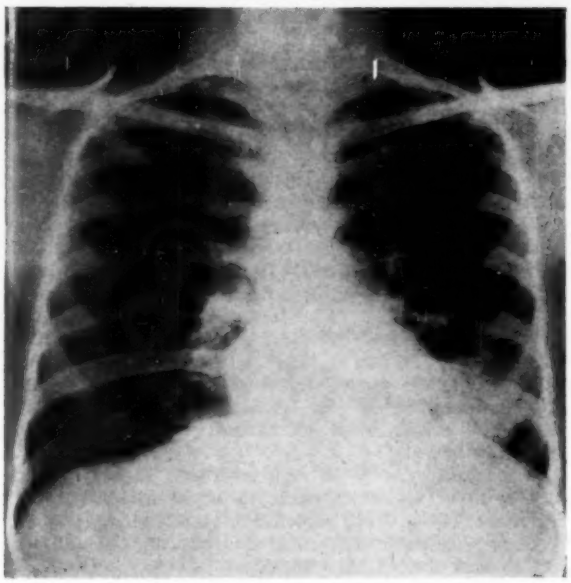

FIG. 774.—Chest radiograph, showing an opacity in the left lower lung field. The azygos vein knuckle is indefinite.

Barium-swallow examination revealed extensive oesophageal varices involving the lower two-thirds of the oesophagus, being most prominent in the lower 2 in. No abnormalities were demonstrable in the stomach or duodenum on barium-meal examination.

Radiographs of the chest showed a persistent shadow in the left lower lung field, identical with the appearances in several chest films taken intermittently over the five months preceding the patient's admission to Manchester Royal Infirmary (*Fig.* 774).

Serological tests for syphilis were negative.

The intradermal Mantoux test, using old tuberculin (1–1000), was negative.

Venous pressure studies were performed, using a vein on the dorsum of the right foot. The resting venous pressure in the erect posture (manubrial height 88 mm. Hg) was 90 mm. of mercury. On slow walking, it fell to 60 mm., and on fast walking to 40 mm. Hg. The venous pressures on exercise were considered abnormally high, and, in the absence of varicose veins, constituted evidence of venous obstruction (Boyd, Catchpole, Jepson, and Rose, 1952).

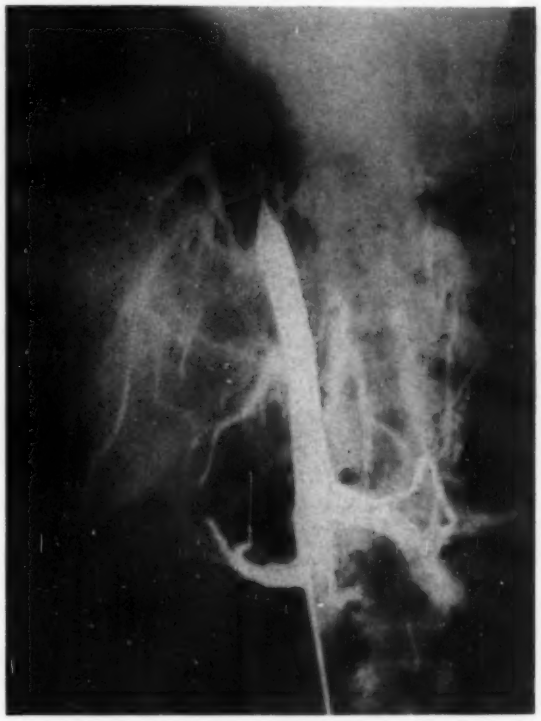

FIG. 775.—Venogram of inferior vena cava. There is a conical block in the inferior vena cava at the level of the 9th dorsal vertebra. The contrast medium also outlines two patent hepatic veins, and a grossly dilated inferior hemiazygos vein, communicating with the left renal vein. The tip of the polythene catheter can be seen in the inferior vena cava at the level of the right renal vein.

In order to determine the level of the inferior vena caval block, a venogram was done; a polythene catheter was passed into the right saphenous vein at the saphenofemoral junction (*see Fig.* 773) for a distance of 11 in., its tip then lying in the inferior vena cava at the level of the right renal vein (X-ray control). Seventy per cent diodrast (40 ml.) were introduced in 15 seconds. Further radiographs showed a conical inferior vena caval block at the level of the ninth dorsal vertebra, and also demonstrated the renal veins, two patent hepatic veins, and an enormously dilated inferior hemiazygos vein clearly outlined by the contrast medium (*Fig.* 775).

Summary: The clinical features and investigations indicated obstruction of the inferior vena cava, with portal hypertension. There was a well-developed collateral circulation, but there was also early hepatic insufficiency.

PROGRESS AND TREATMENT.—On admission, the patient was asymptomatic and remained ambulant while under investigation. Her appetite was good and there were no further hæmatemeses. The hepatosplenomegaly remained unchanged. The skin condition was never

finally diagnosed, and was treated by application of zinc and ichthyol cream.

About seven weeks after admission slight pitting œdema began to appear around the ankles after she had been up for a few hours, and signs of early ascites were noted for the first time. These observations, considered in conjunction with signs of early hepatic insufficiency, indicated the necessity to attempt surgical relief of the inferior vena caval obstruction. Although there seemed little prospect of relieving caval obstruction due to thrombosis, the venographic appearance of the block at the level of the diaphragm suggested the possibility of a congenital valvular obstruction, as described by Bennett (1950), or an unduly narrow caval hiatus in the diaphragm. With this in mind, it was hoped that surgery might establish an improved venous drainage of the liver, and retard the march of events.

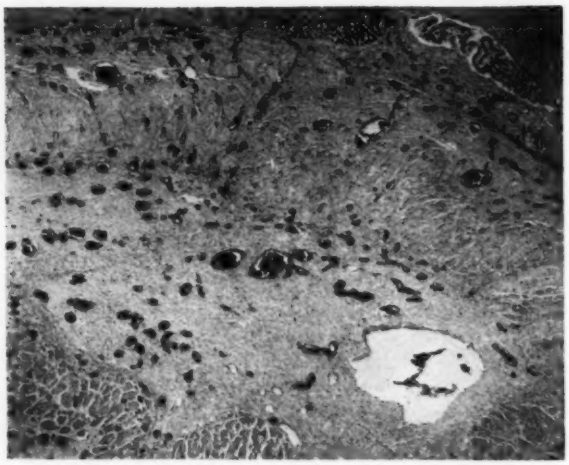

FIG. 776.—Photomicrograph of section of inferior vena cava. The architecture of the inferior vena cava is fairly well preserved. The lumen has been recanalized by numerous vascular channels, which also permeate the adventitia. (× 27.)

OPERATIVE PROCEDURE.—On Oct. 13, 1952, a thoracolaparotomy was performed by Mr. W. F. Nicholson, under general anæsthesia. With the patient in the left lateral position, an incision was made along the line of the 8th rib, extending from the mid-clavicular to the mid-scapular line. Dissection entailed the ligation of many collateral veins in the chest wall. The thorax was entered after removal of the 8th rib and division of the 7th costal cartilage. There were many more grossly dilated veins in the parietal and diaphragmatic pleura.

The mediastinal pleura and pericardium were opened, the latter containing some excess fluid. The thoracic portion of the inferior vena cava thus displayed was found collapsed but apparently patent, and was traced down to a narrow opening in the diaphragm.

The aponeurosis of the diaphragm was transected transversely, revealing an irregular, 'hob-nailed' liver surface. After dissection of the diaphragm from the bare area of the liver, the diaphragm was further divided along a line from the original transverse incision to the narrowed caval hiatus. The inferior vena cava immediately below the diaphragm was then palpated; a fibrous cord could be felt in it posteriorly, which became thicker at the same time as the vein became narrower, progressing downwards into the liver substance.

Since the caval stenosis was found to be situated within the liver substance, it was decided to abandon the operation.

One pint of blood was transfused during the operation. The patient's condition on return to the ward was fair.

Death took place six hours post-operatively; though she regained consciousness, her airway could not be completely cleared of much frothy mucus, so that she became progressively more cyanosed and dyspnœic.

ABSTRACT OF AUTOPSY RECORD (Dr. J. P. Smith).—

Skull and brain: No significant abnormality.

Larynx and œsophagus: Healthy, and no evidence of œsophageal varices, though at one point in the lower end of the œsophagus there was a firm, raised, irregular white area which may have represented an old thrombosed varix.

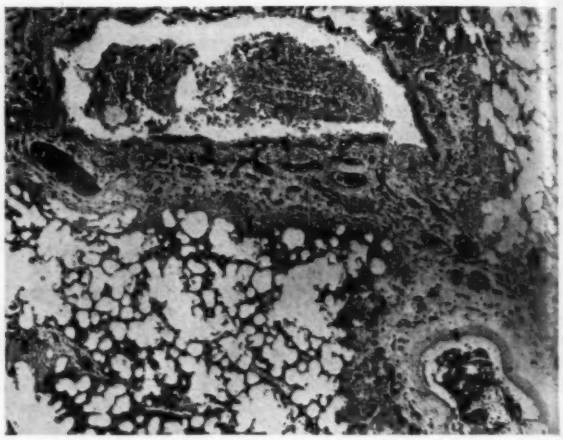

FIG. 777.—Photomicrograph of a section of lung. Cross-sections of a bronchus and of a vessel are shown, with numerous capillary channels occupying the peribronchial and perivascular tissues. (× 27.)

Bronchi and trachea: Intensely congested, containing heavily blood-stained mucus.

Thorax: The right pleural cavity contained a little blood-stained fluid. The pleural surface of the right lung was covered with scanty fibrinous exudate, especially over the costophrenic angle near the operation site, where this was quite thick. The left pleural cavity contained no excess of fluid and the pleural surfaces were healthy.

Lungs: Right 500 g., left 450 g. Both slightly congested and œdematous; cut surface showed a peculiar, irregular mottling of pale areas and dark-red coloured areas. No evidence of thrombosis or emboli in the pulmonary vessels.

Pericardial cavity: Apart from some fibrinous exudate, there was no significant abnormality.

Heart: 180 g. No significant abnormality.

Aorta: Some fatty intimal streaking in both the thoracic and abdominal portions.

Azygos veins: Dilated and tortuous, showing some anastomotic channels.

Inferior vena cava: Ended in a tapering fashion 2 cm. cephalad to the entry into it of two large hepatic veins from the left lobe of the liver. From this tapering upper extremity there extended a mass of fibrous tissue for a distance of 2 cm., which passed through the diaphragm, on the upper surface of which it joined a normal length of inferior vena cava just below the right auricle of the heart. This latter portion of the cava appeared normal in size and position, and one or two small veins, 2 mm. in diameter, opened into it from the diaphragmatic muscle.

Abdomen: The peritoneal cavity contained a slight excess of clear yellow fluid. The liver (1550 g.) showed a grossly nodular surface with individual granules up to 3–4 mm. in diameter, with occasional deeper scars. Cut surface showed a fine, nodular pattern, the individual nodules being 3–4 mm. in diameter. The bile-ducts were patent.

The spleen (650 g.) was considerably enlarged; surface was smooth, cut surface showed slight congestion with prominent Malpighian corpuscles.

Ovaries: Rather fibrous, and contained large numbers of cysts.

The gall-bladder, pancreas, adrenals, kidneys and renal pelves, ureters and bladder, uterus, and abdominal lymph-nodes showed no significant abnormality.

Histology.—

Inferior vena cava: The obliterated portion was sectioned serially. Sections showed that the lumen of the vein was replaced by a moderately cellular connective tissue through which ran large numbers of capillary channels. The original muscular wall of the vein could be traced throughout; the muscle-bundles showed some degeneration, and there was increased connective tissue amongst them. The adventitia was considerably increased and blended with the fibrous tissue of the surrounding liver. A rather remarkable feature was the presence in the adventitia of enormous numbers of capillaries (*Fig.* 776).

Serial sections through the blocked area revealed two large hepatic veins which were obliterated by an identical process. Serial sections through one of the small veins draining into the patent portion of the inferior vena cava above the diaphragm showed that the vein entered liver substance and was surrounded by a thickened adventitia permeated by an enormous number of capillaries, as in the main lesion.

Liver: Diffuse cirrhosis in all areas—islands of liver-cells of varying size surrounded by dense cellular fibrous tissue in which large numbers of proliferating bile-ducts were found. In many of the accumulations of fibrous tissue enormously dilated lymphatic channels could be seen.

Lungs: The patchy appearances of the gross specimen were seen histologically to consist of alternate areas of normal lung alveoli and areas of collapsed alveoli which were œdematous and congested. The bronchi showed very gross congestion and hæmorrhages into the mucous membrane. An unusual feature was the presence of large amounts of perivascular and peribronchial fibrous tissue proliferation, heavily permeated by new vascular channels (*Fig.* 777).

Adrenals: In the hilum of one of the adrenals there was a thrombosed vein showing organization, the age of which was estimated to be at least six days.

Skin: There was a mild, patchy perikeratosis with infiltration of histiocytes around sweat-ducts and glands, hair follicles, and vascular bundles in the dermis. These appearances were entirely non-specific.

Brain and meninges: No abnormality was found in the brain substance, but at one point there was some fibrous thickening of the pia-arachnoid over a sulcus, suggestive of a healed meningitis.

DISCUSSION

From an examination of previous literature (Thompson and Turnbull, 1912; Hutchison and Simpson, 1930; Armstrong and Carnes, 1944; Brontë-Stewart and Goetz, 1952), and a careful study of the case reported here, it would appear that the Budd-Chiari syndrome may present as two distinct clinico-pathological types:—

Type A, in which thrombosis commences in the hepatic veins. When most of these are occluded, the patient becomes acutely ill. Its clinical features include: (*a*) Sudden onset of obscure abdominal pain, sometimes extending to the back, with nausea, vomiting, and shock; (*b*) Development of ascites, with tender enlargement of the liver and spleen; and (*c*) After preliminary delirium, a terminal coma leading to death within a few days.

The thrombotic process may extend sufficiently to involve the inferior vena cava at its junction with the hepatic veins. This is of little clinical interest since there is rarely enough time for the signs of inferior vena caval obstruction (*see below*) to develop.

Type B, which is of much greater clinical interest than Type A. The presenting picture is caused by thrombosis of the intrahepatic inferior vena cava. However, it is possible that the process first commences in, and for a time remains limited to, some of the hepatic veins without producing any symptoms whatsoever (Armstrong and Carnes, 1944; Kelsey and Comfort, 1945; Thompson, 1947). The clinical features of this type of case are more gradual in onset and include: (*a*) indigestion, with epigastric discomfort; (*b*) hæmatemesis; (*c*) tender enlargement of liver and spleen; and, most strikingly diagnostic of all, (*d*) the development of obvious subcutaneous venous collaterals over the upper part of the abdomen and lower part of the thorax, where anastomosis between the superior epigastric, medial xiphoid, and internal mammary veins takes place. A true caput medusæ is very rarely seen (Thompson, 1947).

Venography of the inferior vena cava demonstrates a much enlarged azygos venous system acting as a by-pass to the caval block (Brontë-Stewart and Goetz, 1952). Survival is possible for several years, but it may be cut short by a severe hæmatemesis, by mesenteric thrombosis (Kelsey and Comfort, 1945) or by acute liver failure (*see above*) caused by the spread of the thrombotic process to most or all of the hepatic veins (Hoover, 1920; Thompson, 1947). In the absence of any of these contingencies the expectation of life is apt to be diminished by progressive chronic liver failure, i.e., cirrhosis (Hirsh and Manchester, 1946). (*Fig.* 778.)

It will be seen that our patient was suffering from the chronic type (Type B) of the Budd-Chiari syndrome. She presented clinical evidence of inferior vena caval block—in particular, prominent superficial veins in the flanks and back; this, taken in conjunction with evidence of portal hypertension, suggested a diagnosis of Budd-Chiari syndrome. This was confirmed by a venogram of the inferior vena cava, which showed (*a*) a block in the intrahepatic portion of the cava, (*b*) two patent hepatic veins, and (*c*) a big inferior hemiazygos vein that appeared to anastomose with the left renal vein and constituted the main intra-abdominal by-pass of the caval block (*Fig.* 775). The venogram explained why the patient had failed to give a history of any acute (Type A) episode. It demonstrated that a good collateral circulation had become established, suggesting that the date of onset of the disease process was not recent.

The precise anatomy of the paravertebral venous collaterals is of interest in this case. While the venogram of the inferior vena cava showed a big, tortuous inferior hemiazygos vein, a whole series of chest radiographs, including some more penetrating ones, failed to demonstrate any appreciable increase in size of the azygos vein knuckle, at the point where it hooks around the right bronchus to join the superior vena cava (beautifully demonstrated by the case of Brontë-Stewart and Goetz, 1952). The assumption was, therefore, that the inferior

hemiazygos vein did not drain normally into the azygos vein.

The post-mortem histological finding of great numbers of peribronchial vascular channels throughout both lungs may have been of significance in this connexion. Recent work has shown the importance of the bronchial veins as connecting links between the pulmonary and azygos venous systems in diseases

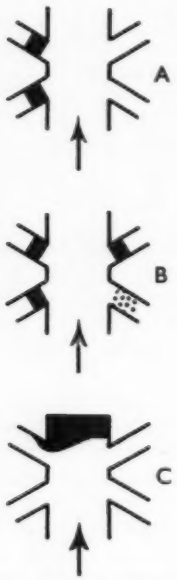

FIG. 778.—Diagram relating extent of thrombosis at junction of hepatic veins with inferior vena cava to clinical features of Budd-Chiari syndrome. A, Only two hepatic veins are occluded by thrombosis—no Budd-Chiari syndrome; B, All hepatic veins are blocked by thrombosis—Type A of Budd-Chiari syndrome; C, Inferior vena cava above junction with hepatic veins blocked by thrombosis—Type B of Budd-Chiari syndrome.

associated with pulmonary venous hypertension (Tobin, 1952). In our patient there was undoubtedly considerable azygos venous hypertension, and it is therefore possible that the well-developed peribronchial channels were in fact carrying blood from the azygos system to the pulmonary veins. This supposition, in turn, might explain the patient's asthma, and might even label the persistent shadow in the left lower lung field (*Fig.* 774) as an area where the bronchial veins draining the inferior hemiazygos vein were particularly large and numerous.

These considerations we consider to have a direct bearing on the cause of death of our patient. At operation, many of the big collateral vessels in the body wall were unavoidably cut, and this was inevitably followed by an increased flow of blood through the remaining channels. The intense congestion of the bronchi, the submucous hæmorrhages, and the presence of large quantities of hæmorrhagic mucus in the trachea, bronchi, and alveoli suggested that the increased load was too heavy and was the direct cause of death.

Whatever the exact explanation, surgical intervention in the treatment of this syndrome almost always accelerates death. Thus, Thompson (1947) states that, of 9 patients operated on, 8 died, the majority within 2–3 days, and 2 within twelve hours (also Armstrong and Carnes, 1944). In view of the fair prognosis carried by the chronic form of the Budd-Chiari syndrome (Type B), it is of paramount importance that patients be properly investigated—by barium swallow, liver function tests, and venography—as only in this way can the diagnosis be substantiated, and recourse to surgery, whether for diagnostic or therapeutic purposes, avoided.

It appears, therefore, that there is good reason for widening the definition of the Budd-Chiari syndrome, and to take note not only of the pathological, but also of the clinical, features, as demonstrated by this case. The implications of this have, perhaps, not received adequate consideration in the past.

SUMMARY

A case of the Budd-Chiari syndrome is described. The syndrome has been separated into two distinct clinico-pathological types :—

Type A.—This is caused by thrombosis of most, or all, of the hepatic veins, leading to acute liver failure and death within a few days.

Type B.—Here, thrombosis of the inferior vena cava gives rise to warning manifestations of the disease, which usually runs a chronic course of some years.

The importance of the collateral venous channels in this case is discussed, and the significance in relation to the fatal consequences of surgical intervention outlined.

It is emphasized that every endeavour should be made to arrive at a correct diagnosis clinically. The undesirability of surgical intervention is noted.

We are grateful to Dr. J. F. Wilkinson for the interest he has taken in the preparation of this paper, and for his valuable advice and criticism. We also wish to thank Mr. W. F. Nicholson for his help and guidance.

Mr. R. P. Jepson, Lecturer in Surgery, University of Manchester, kindly performed the venogram of the inferior vena cava.

Dr. R. G. W. Ollerenshaw, of the Department of Clinical Photography, Manchester Royal Infirmary, and Mr. S. G. Heaton are thanked for their help in the preparation of the illustrations.

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AN EVALUATION OF VENOGRAPHY AND VENOUS PRESSURES IN THE STUDY OF THE LEG VEINS

By H. D. MOORE

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ASCENDING and descending venography and the venous pressure in the feet are used to study the veins of the legs.

It has been stated that *ascending venography* will always show the deep veins of the legs (except the anterior tibials), their size, outline, and valves (when present), and the patency of the femoral and iliac veins (Moore, 1949).

Descending venography is said to outline the valves of the femoral veins, when present (Bauer, 1948), but statements differ about the distance the diodone will descend; Bauer states that it is arrested in the upper third of the thigh when the valves are adequate, but descends into the legs when they are not. Luke (1941) and Dow (1951) claim, however, that the diodone will descend into the legs in the majority of normal persons.

Venous pressure readings have demonstrated that, in any leg, the pressure in the veins of the feet, when standing motionless, equals the height of a column of blood extending from the heart to the point of measurement. In normal legs, upon movement (i.e., standing/walking) the pressure drops sharply (Smirk, 1935), whereas in the gravitational syndrome it is stated that the pressure, upon movement, falls slightly, if at all, and may actually rise (Warren, White, and Belcher, 1949; and others).

FIG. 779.—Ascending venogram of a normal leg using 40 c.c. of 35 per cent diodone. The anteroposterior view of the leg shows anterior tibial and peroneal venæ comites with frequent bulgings at the site of the valves. The muscular vessels are seen and the popliteal vein is double. The lateral film shows anterior tibial, peroneal, and posterior tibial veins with multiple valves. The muscular vessels are in two groups. The popliteal shows valves (one of which has the typical fish-tail appearance). The exposure for the thigh and knee shows the union of the popliteal veins to form a femoral of normal size, but in which the valves are only vaguely shown.

FIG. 780.—Descending venogram using 20 c.c. of 35 per cent diodone and the patient inclined, feet down, at an angle of 45°, to show the medium held mainly at the first valve and finally at the second. The leg was symptomless and the venous pressures were 70, 12, and 8 mm. Hg respectively.

These three procedures have never been correlated, and it was decided, therefore, to carry out ascending and descending venography, and pressure readings in the veins of the feet to see if the results of the three procedures agreed, and to try and assess the value of venography in the legs, as a whole, and the relative values of the ascending and descending methods.

METHOD

1. The method of ascending venography was that of Moore (1949). In brief, this consists of injecting diodone into any vein on the foot of the supine patient; using a tourniquet at the ankle to occlude the superficial veins and to force the diodone

into the deep venous system; and applying a tourniquet to the thigh to occlude both the venous return and the diastolic inflow of arterial blood.

By this method the deep veins are well seen, their valves are outlined, and the muscular vessels fill (*Fig.* 779).

2. The method of descending venography consisted of a 'blind' puncture of the femoral vein with the needle pointing towards the head of the patient. When the vein was entered, an assistant compressed it above the needle against the pubic ramus, and 20 c.c. of 35 per cent diodone were injected. It has been my experience that the femoral vein needs to be occluded proximal to the site of injection to produce consistently good filling of the veins. On the other hand, the patient can be supine or erect without making the slightest difference to the results. (*Figs.* 779, 780 A, 781.)

3. The method of venous pressure readings was that of Walker and Longland (1950). This consists of putting a polythene tube into one of the veins of the foot, and connecting it with a reservoir of saline or heparinized saline, a manometer, and an air bulb to raise or lower the pressure. The venous pressure is taken to be that pressure at which the blood is prevented from proceeding along the polythene tube. The end-point for the readings is not very definite, so that they are not strictly accurate; but, on the other hand, a series of readings in the same patient approximate quite closely, so that they are accurate enough —especially as the figures of interest are those showing the changes, rather than the absolute pressures.

The pressures taken were the standing pressure; the pressures when the patient was standing/walking, lifting the feet about 18 in. 30–40 times per minute; and the pressures standing/walking with a tourniquet below the knee tight enough to occlude the superficial, but not the deep veins.

RESULTS

The Findings in 21 Legs which were without Symptoms except for Superficial Varicose Veins.—In the *descending venograms* the outline of the veins was smooth and regular, the vessels were of normal size, and valves were well marked in all the legs except one, where they were poorly defined (*Table II, Case* 5, left leg). The diodone was sometimes sharply arrested at the first or second valve in the femoral vein (*Fig.* 780), but as often descended as far as the knee, despite the presence of several normal looking valves (*Fig.* 781 A); and, frequently the diodone would be held at the first or second valve on one side but descend to the popliteal region on the other, despite normal looking valves and despite the fact that the venous pressures were almost the same on each side and showed a considerable fall with exercise (*Fig.* 781 A). In several cases the diodone descended to the popliteal region and then tailed off, giving the impression that it was not held at a valve but that there was insufficient diodone to

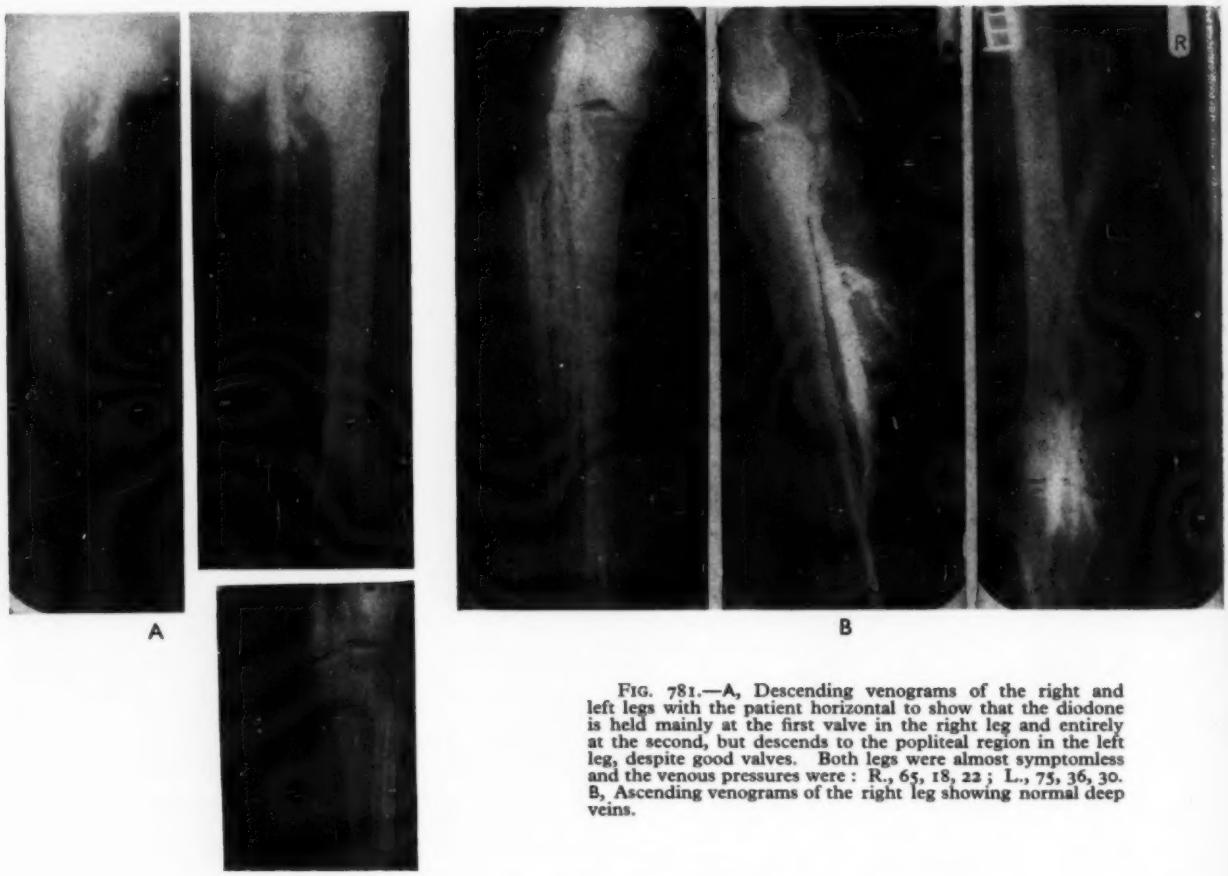


FIG. 781.—A, Descending venograms of the right and left legs with the patient horizontal to show that the diodone is held mainly at the first valve in the right leg and entirely at the second, but descends to the popliteal region in the left leg, despite good valves. Both legs were almost symptomless and the venous pressures were: R., 65, 18, 22; L., 75, 36, 30. B, Ascending venograms of the right leg showing normal deep veins.

outline the veins (*Fig.* 781 A); and in one case the diodone descended to the calf, outlining several normal looking valves (*Fig.* 782) (in all these cases the venous pressures showed a considerable fall on exercise).

The *ascending venograms* (*Fig.* 781 B) showed vessels of normal size and outline and valves were

FIG. 782.—Descending venogram using 20 c.c. of 35 per cent diodone and the patient tilted, feet down, 20° from the horizontal. The veins are outlined to mid-calf despite obvious valves all the way down, symptomless legs, and venous pressures of 78, 40, 28.

demonstrated in all but one. The valves were not, however, always well shown and their presence or absence was arguable in several.

The *venous pressures* (*Table I*) showed an average fall, with standing/walking, of 43 mm. Hg, and with a tourniquet below knee of 51 mm. Hg. The fall was sometimes a good deal more, and even the least fall was 16 and 26 mm. Hg respectively. The increased fall of pressure on standing/walking, when a tourniquet was placed below the knee, is presumably due to the fact that the valves of the superficial veins were incompetent—these patients, of course, had superficial varicose veins, and where the diodone

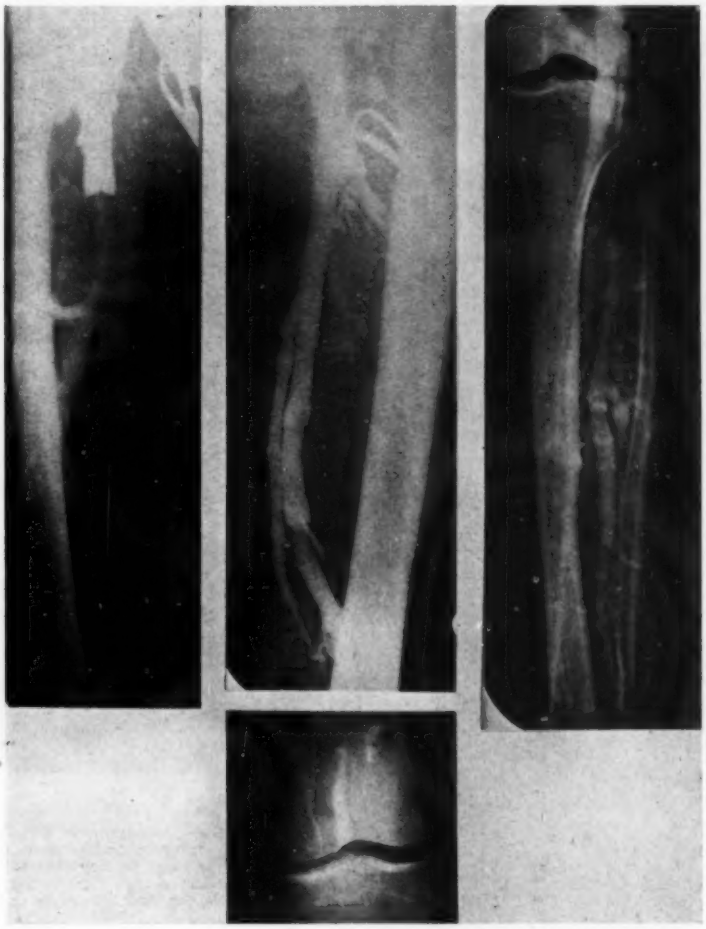


FIG. 783.—Descending venograms, right and left sides, and ascending venogram left, of *Case* 1, *Table II*. The diodone is held mainly at the first valve on the right side, but descends to the popliteal region on the left, although the symptoms were more marked on the right. The ascending venogram is normal and all three show good valves. The venous pressures were: R., 88, 42, 35; L., 88, 40, 40.

entered the superficial veins it descended without let or hindrance (*Fig.* 781 A). Whether this group would go on to the stage of the next group (*Table II*) can only be decided by waiting for years to see.

There are, unfortunately, no true normals for comparison, for it was not possible to persuade any person with completely normal legs to have two venograms and a pressure reading done.

The Findings in 9 Legs with Symptoms more or less typical of the Gravitational Syndrome which were relieved of their symptoms by ligation of the superficial veins alone or in conjunction with mild support in the form of elastic stockings.

None of these patients had any history suggestive of a deep venous thrombosis in the leg in question. Their symptoms are briefly given in *Table II* in

which the ascending and descending venograms and venous pressures are compared with the normal side (when only one side was affected).

In these 9 legs the average fall in the *venous pressure* was 35 and 42 mm. Hg, respectively but in 1 leg (*Case* 7) the fall was only 30 and 15 mm. Hg.

The *descending venograms* showed vessels of normal outline with good valves in 7 legs (*Fig.* 783). In *Case* 6 the outlines were irregular and incomplete. In *Case* 7 the outline was normal, but the valves were poorly defined.

The *ascending venograms* showed vessels of normal size and outline in 8 legs (*Fig.* 783), but in *Case* 4 the vessels were rather large. The valves were demonstrated in all the cases, but were difficult to see in *Cases* 4, 5, and 7.

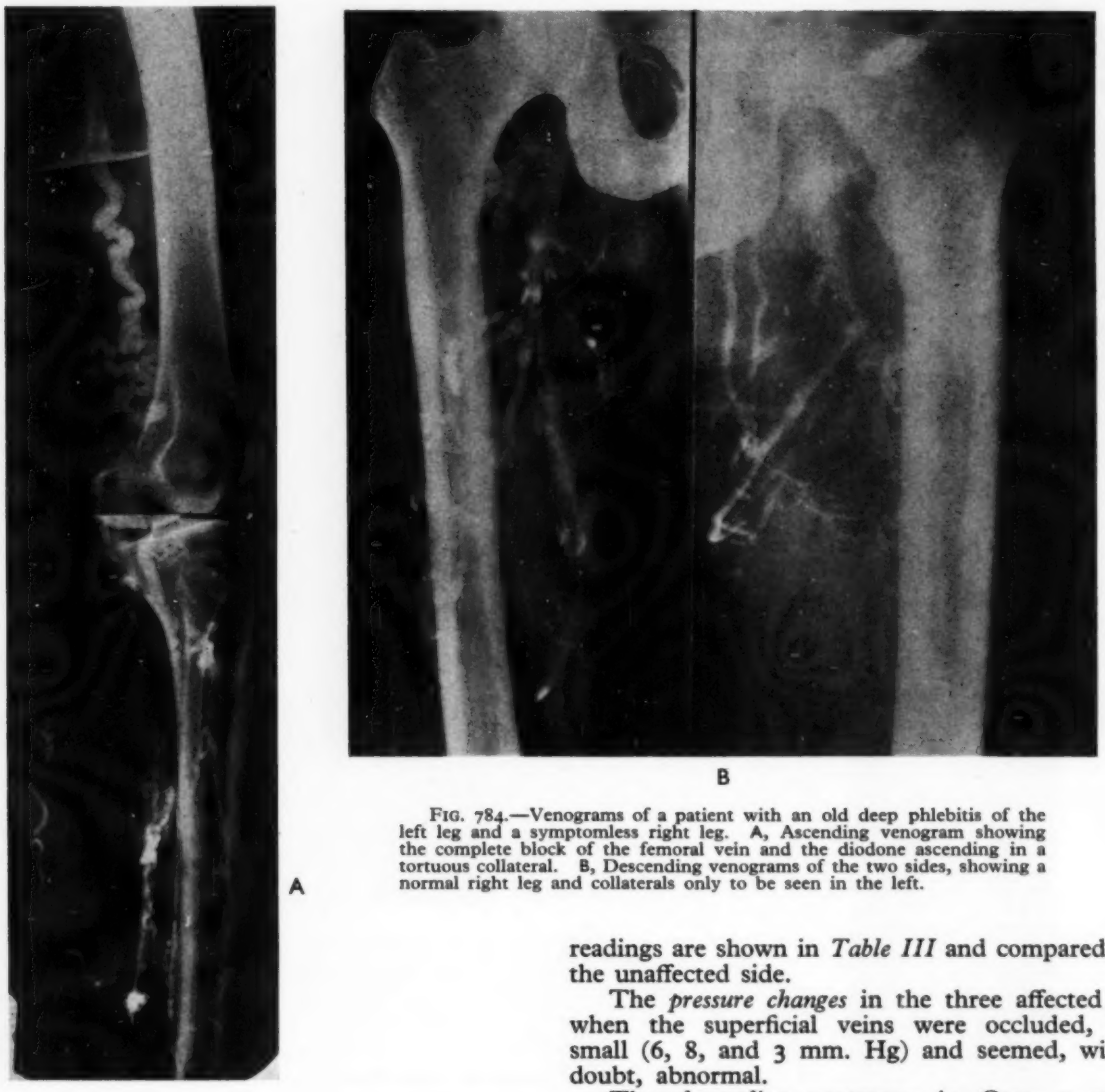
The results are, therefore, that :—

In *Cases* 1, 2, and 3 (5 legs) the three tests agreed and were normal.

In *Cases* 4, 5, and 6 (3 legs) two tests were unequivocally normal and agreed and settled the doubt about the third test.

In *Case* 7 (1 leg) none of the tests were unequivocal, but the three taken together suggested normal deep veins—a conclusion which was supported by the response to treatment.

The Findings in 3 Legs with Symptoms of the Gravitational Syndrome and with a History of a Previous Deep Venous Thrombosis.—The symptoms, venographic findings, and pressure readings are shown in *Table III* and compared with the unaffected side.



A B

FIG. 784.—Venograms of a patient with an old deep phlebitis of the left leg and a symptomless right leg. A, Ascending venogram showing the complete block of the femoral vein and the diodone ascending in a tortuous collateral. B, Descending venograms of the two sides, showing a normal right leg and collaterals only to be seen in the left.

The *pressure changes* in the three affected legs, when the superficial veins were occluded, were small (6, 8, and 3 mm. Hg) and seemed, without doubt, abnormal.

The *descending venograms* in *Cases* 1 and 2 showed a few collaterals only (*Fig.* 784 B) and were probably abnormal, but the findings, if considered alone, may have been the result of a technical error. *Case* 3 showed a femoral vein of normal outline, but no valves were demonstrated (*Fig.* 785). The vessel was, therefore, probably abnormal, but not unequivocally so.

The *ascending venograms* in *Cases* 1 and 2 showed a complete block of the femoral veins, with the diodone ascending in the collaterals (*Fig.* 784 A). The venogram in *Case* 3 was not obviously abnormal.

The results, therefore, are that : In *Cases* 1 and 2, the three tests agreed and showed that the deep veins

were abnormal. The ascending venogram, however, was the only test which showed, without doubt, the site and nature of the abnormality. In *Case* 3, the venograms were equivocal, but taken in conjunction with the venous pressures, show abnormality of the deep veins. In this case the ascending venogram was misleading and would have led to error if taken alone.

CONCLUSIONS

The ascending and descending venograms and venous pressures usually agree (in this series in 28 out of 33 legs). If any one of the three examinations shows an unequivocal normal, then that finding can be taken as valid; but if a finding is abnormal or if its normality is arguable, then it is most unwise to rely on one test alone.

The venous pressures appear to be the most reliable index of the state of the veins. When the veins are normal the changes in pressure, however, are sometimes not great enough to be sure of normality without further evidence (*Tables I* and *II*) and when the veins are abnormal, the pressures do not indicate the nature of the abnormality.

Table I.—Venous Pressures in 21 Legs which were Symptomless apart from Minor Varicose Veins

Case No.	Leg	Pressure Readings in mm. Hg			Referred to in
		Standing	Standing/walking	Standing/walking with tourniquet	
1	L.	80	45	22	*Table II, Case* 5
2	R.	78	50	30	
	L.	76	60	50	
3	R.	76	20	50	*Table III, Case* 1
4	R.	90	38	20	*Table II, Case* 2
5	R.	80	15	0	
	L.	78	40	28	
6	R.	80	32	20	
	L.	75	18	22	
7	R.	75	36	30	
	L.	65	18	22	
8	R.	80	42	30	
	L.	75	36	34	
9	R.	78	28	Not done	
	L.	78	46	Not done	
10	R.	75	44	28	*Table II, Case* 4
11	R.	88	35	20	
	L.	88	32	15	
12	R.	70	12	8	
	L.	70	20	14	
13	L.	77	61	48	*Table III, Case* 2

Average fall of pressures: Standing/walking, 43 mm. Hg; Standing/walking with tourniquet, 51 mm. Hg.

Descending venography is the only method which will show the valves in the femoral vein, but failure to outline the vein is as likely to be a technical fault as a fault in the vessel—therefore a positive finding is most valuable, but a negative one must be disregarded.

Ascending venography is the only way to outline the veins of the legs and their valves, and the veins of the thigh when the femoral vein is blocked (*Table III*). It is easy to do, and filling of the deep veins can be relied upon, but the interpretation of the films can be difficult and may lead to error if used alone.

It would seem, therefore, that venography should not be the first approach to the study of the veins of the legs, but that it should be used in all cases where the venous pressure readings show a definite abnormality or are doubtful—and that ascending venography should be used before descending, the descending method being reserved for those cases which are not clear after the venous pressures have been taken and ascending venograms made.

Fig. 785.—Ascending and descending venograms of a patient with ulcers and venous pressure of 87, 88, 84, showing considerable bulging of the femoral vein at what is presumed to have been the site of the valves, but no outline of the valves themselves in the main vein (*Table III, Case* 3, right leg).

A point of some interest which also emerges from this study is that the symptoms of the gravitational syndrome (that is to say aching of the legs, a brownish discoloration and induration of the front (sometimes the whole) of the lower leg, with swelling of the ankles towards the end of the day, and ulceration in the malleolar region or front of the lower third of the limb) can be produced by faults in the superficial veins only. This is well shown in *Table II*, and is not only of interest, but of importance in that an operation on the superficial veins will cure the patients.

Table II.—FINDINGS IN 9 LEGS WITH SYMPTOMS OF GRAVITATIONAL SYNDROME

CASE AND LEG WITH SYMPTOMS	SYMPTOMS	LEG	ASCENDING VENOGRAM	DESCENDING VENOGRAM	PRESSURE READINGS IN MM. Hg		
					Standing	Standing/ walking	Standing/ walking with tourniquet
1. Both legs	Varicose veins *both legs*. Aching of calves and swelling of ankles, both legs. 'Eczema' R. leg. Bilateral Trendelenburg operation with relief of symptoms	R.	Vessels normal size. Valves shown	Diodone descends to popliteal region. Good valves	88	42	35
		L.	Ditto	Diodone held upper third thigh. Good valves	88	40	40
2. L. leg	*R. leg symptomless*	R.	Ditto	Descent to upper third of leg. Valves good	90	38	20
	Varicose veins and an ulcer of *L. lower leg*. Relieved by support	L.	Ditto	Descends to popliteal region. Valves good	90	35	45
3. Both legs	Varicose veins and swelling of ankles *both legs*. Some aching. 'Eczema' round R. ankle. Relief with Trendelenburg operation	R.	Ditto	Diodone held mainly in upper third of thigh, but some descends to popliteal region. Good valves	78	46	36
		L.	Ditto	Ditto	78	38	27
4. L. leg	*R. leg symptomless*	R.	Vessels rather large. Valves shown	Diodone held in upper thigh. Good valves	75	44	28
	Varicose veins and 'eczema' *L. leg*. Relieved by Trendelenburg operation	L.	Vessels rather large. Valves difficult to see but present	Ditto	75	60	30
5. R. leg	Varicose veins *R. leg*, and ulceration and discoloration of R. shin	R.	Vessels normal size. Valves present but difficult to see	Diodone held in upper part of thigh. Valves good	80	50	35
	Varicose veins but *no symptoms L. leg*	L.	Ditto	Diodone descends to upper leg. Valves poor	80	45	22
	Complete relief with Trendelenburg operation on R. veins, and support						
6. L. leg	(Ulceration and induration of *R. leg*, *Case* 3, *Table III*)	R.	—	*See Table III*	—	—	—
	Left leg: some œdema and a few varicose veins. Otherwise normal	L.	Vessels normal size. Valves present	No valves seen. Outline irregular and incomplete. Diodone descends to knee	85	62	50
7. Both legs	Varicose veins and swelling of ankles of *both legs*—more on R. Relief with operation on superficial veins, plus support to veins	R.	As for *Case* 5	Diodone descends to upper leg. Valves present but poor	80	50	65
		L.	Not done	Not done	Not done	Not done	Not done

Average fall of pressure: Standing/walking, 35 mm. Hg; Standing/walking with tourniquet, 42 mm. Hg

Table III.—FINDINGS IN 3 LEGS WITH SYMPTOMS OF GRAVITATIONAL SYNDROME AND HISTORY OF DEEP VENOUS THROMBOSIS

CASE NO. AND LEG AFFECTED	SYMPTOMS	LEG	ASCENDING VENOGRAM	DESCENDING VENOGRAM	PRESSURE READINGS IN MM. Hg		
					Standing	Standing/ walking	Standing/ walking and tourniquet
1. L. leg	R. leg symptomless	R.	Vessels normal size and valves present	Diodone held mid-thigh. Valves good	76	20	50
	L. leg swelling and aching, and large varicose veins	L.	Vessels normal size in leg; complete block of femoral; blood returns in large collaterals. No valves seen	Only a few collaterals seen	*84*	*64*	*78*
2. L. leg	R. leg symptomless, with a few varicose veins	R.	Vessels normal size and valves present	Diodone descends to knee, but the greater part held above the first valve	77	61	48
	L. leg ulceration and induration	L.	Vessels rather small and irregular. No valves seen. Femoral vein thrombosed and blood returns in a plexus in the line of the vein	Only a very few collaterals seen	*87*	*81*	*79*
3. R. leg	R. leg ulceration and induration	R.	No obvious abnormalities. Some valves seen	Valve bulgings seen, but no valve outline. Diodone descends to lower thigh	*87*	*88*	*84*
	L. leg: some œdema and a few varicose veins; otherwise normal	L.	Normal size. Valves present. Femoral vein normal	No valves seen. Outline irregular and incomplete. Diodone descends to knee	85	62	50

SUMMARY

Thirty-three legs were examined by ascending and descending venography, and by taking the venous pressures in the feet when standing still, when standing/walking, and when standing/walking with a tourniquet below knee to occlude the superficial veins.

Twenty-one legs were symptomless, apart from minor varicose veins; 9 had symptoms of the gravitational syndrome, but no history of phlebitis and a good response to ligation of the superficial vein; and 3 had symptoms and a history of a deep phlebitis.

It was found that the results of the three tests usually agreed; that an unequivocal normal with any test could be relied upon; but that a doubtful result or an abnormal reading required further investigation.

It was concluded that the venous pressure readings were the most reliable test of the state of the deep veins. If the readings were normal, no further investigation was necessary, but if they were abnormal or doubtful, then ascending venography should follow, descending venography being done only in the few cases where doubt still existed.

It was shown that the symptoms of the gravitational syndrome can be produced even when the deep veins are normal, by faults in the superficial veins.

It gives me great pleasure to thank Dr. J. T. Ingram for referring *Case* 5, *Table II*, for study; Dr. S. Anning for *Case* 6, *Table II*; and Professor P. J. Moir for permission to examine the remainder.

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PRIMARY SKIN-GRAFTING IN PROCTOLOGICAL SURGERY

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ANO-RECTAL surgery is so heavily contaminated by such a wide variety of organisms that the surgeon has always been reluctant to provide a medium which might allow these bacteria to infect the tissues. The surgeon's attitude to wounds in the area has been one of conservatism despite the experience gained in the treatment of the open wound in two world wars and the availability of an increasing range of effective antibiotic agents.

It has been, and in many clinics it is still, the standard practice to allow wounds in the region to heal by granulation tissue. The wound is covered with a bland dressing; macroscopical granulation tissue appears between the tenth and fourteenth days and at the same time a thin layer of epithelium can be seen growing inwards from the edge of the wound. The wound is contaminated but bacterial invasion of the deeper tissues is rare because there is no suitable static culture medium present and the granulation tissue provides a satisfactory 'barrier'. This method of healing by 'secondary intention' has disadvantages. The surgeon must watch the wound closely in the post-operative period to prevent the formation of pockets that may harbour organisms long after the epithelium has covered the wound; such close observation is rendered the more difficult in this area by the upward extension of the wound into the anal canal, which makes the upper half of the wound difficult to see and control. The tendency of granulating wounds to contract constitutes a serious disadvantage in the treatment of post-operative anal stenosis and fissure-in-ano. The surgeon is bound, therefore, to make a special study of the wound he makes in this area until it has healed, and this entails a period of hospitalization and convalescence which tends to be out of proportion to the seriousness of the lesion for which the patient sought advice.

Recognition of these disadvantages has led surgeons to attempt primary suture of ano-rectal wounds. It is difficult to secure perfect hæmostasis prior to wound closure and impossible afterwards to apply firm dressings to close any smaller vessels. Hæmatoma formation is likely, and contamination may well lead to infection and recurrence. Despite speedy convalescence, primary suture has never been popular with proctologists because of the uncertainty of the end-results.

Skin-grafting the granulating wound has been advocated and widely practised (Gabriel, 1927; Wright, 1934). Although the results have proved satisfactory the procedure is not as reliable as skin-grafting fresh wounds; furthermore it entails a second period of enforced bed-rest just at the time the patient is resuming active ambulation.

The obvious solution to these difficulties is primary or delayed primary skin-grafting (Hughes, 1952, 1953, a, b, 1954). The fresh wound is completely covered with a split-skin graft (Thiersch graft). This type of graft will take well on a fresh wound, no matter how contaminated, provided the two surfaces can be maintained in close apposition for several days. Infection will not develop unless the organisms be particularly virulent or unless a suitable medium exists in the form of dead tissue or stagnant pools of tissue fluid. Most wounds in the region are simple wounds; hæmorrhage is readily controlled and the graft can be applied at once and kept in position for the requisite time.

Large wounds present a more difficult problem; hæmorrhage may not be satisfactorily controlled at the initial operation and the surgeon is naturally not prepared to risk the possibility of the skin-graft becoming lifted off the wound by a reactionary bleed in the immediate post-operative period. These wounds should be treated by delayed primary

skin-grafting. The initial wound is gently packed with dry gauze which is left undisturbed until the second or third post-operative day. The patient is then returned to the theatre, re-anæsthetized, and the pack removed. After any blood-clot and debris is irrigated away, the skin-graft is applied. The wound should not be packed with ' Vaseline ' gauze initially as this will tend to leave a smear of ' Vaseline ' over the wound and make it more difficult to obtain accurate apposition of the skin-graft to the wound. The wound should not be left longer than two or three days because after this time the surgeon will have to contend with infection established over the surface of the wound. Moreover, it is desirable to keep the bowels confined for the duration of the treatment, and if the interval between the initial operation and the skin-graft be unduly prolonged the discomfort towards the end of the combined procedure may become intense.

FISSURE-IN-ANO

A fissure-in-ano, usually situated in a dorsal position, is responsible for considerable discomfort to the patient. Acute and subacute fissures may heal with the aid of non-operative treatment, but the chronic fissures with skin and mucosal tags and with fibrotic base are best excised. The subcutaneous sphincter should be divided in the floor of the wound.

Table I.—RESULTS

LESION	NO. OF CASES	PERCENTAGE ' TAKE '			CAUSE OF UNSATISFACTORY RESULT
		90–100	70–80	60 or less	
Fissure-in-ano	14	13	1	—	—
Anal stenosis	3	2	—	1	Avulsion at first dressing
Low anal and uncomplicated high anal fistula	21	16	3	2	Hæmorrhage (1), infection (1)
Ano-rectal fistula	2	1	—	1	Hæmorrhage

Left to heal by granulation tissue the wound is not usually healed in less than four weeks and it may take six weeks. Unless carefully watched there is a tendency for the edges of the wound to fall together and form a pocket. Furthermore the fibrosis deep to the granulating surface tends to cause contraction and may produce a bar of tissue which encourages recurrence.

A consecutive series of 14 patients suffering from chronic dorsal fissure-in-ano were treated by excision of the fissure, division of the subcutaneous sphincter, and primary split-thickness skin-grafting (*Table I*). The first dressing was done on the fourth or fifth day. In 13 instances the ' take ' of the graft was 100 per cent; in 2 of these cases the piece of skin had not completely covered the wound, and in 1 case the graft had shifted slightly to one side during the application. All these patients were discharged from hospital on the fifth or sixth post-operative day with the wound healed or nearly healed and no longer in need of special dressing technique. In the fourteenth case hæmorrhage under the graft was responsible for the loss of 30 per cent of the graft. All these patients have been followed up for a period varying from six to eighteen months, and in no case has there been any recurrence.

ANAL STENOSIS

Stenosis at the anal margin most often follows a faulty hæmorrhoidectomy. Too much skin is removed, and the wounds healing by granulation tissue contract to give a tight anus. With each bowel action the skin tears, to give chronic skin cracks which aggravate the stenosis. These have been treated in the past by excision of a wedge of tissue from the anal margin and allowing the resulting wound to heal by secondary intention.

Anal stenosis is admirably suited to excision and primary skin-grafting. Three cases of anal stenosis, each following hæmorrhoidectomy, have been treated by excision of a wedge of tissue from the anal margin with the immediate application of a skin-graft (*Table I*). In two of these cases the graft showed a 100 per cent ' take ' and the patient left hospital on the sixth post-operative day without requiring further attention. In the third case the dressing was done on the fourth post-operative day; the outer edge of the dressings were lifted up and the graft appeared sound. But the patient, a little apprehensive, suddenly pulled away, and the graft was largely avulsed from its bed. The wound was then allowed to heal by secondary intention; five weeks elapsed before the wound was completely healed. Twelve months, nine months, and six months respectively have elapsed since the operations without signs of recurrence.

LOW ANAL AND UNCOMPLICATED HIGH ANAL FISTULA-IN-ANO

The most common type of fistula-in-ano is the low anal fistula which is difficult to separate from the uncomplicated high anal fistula. In this series of fistulæ 21 were of this variety and 2 were ano-rectal. The track of the fistula is superficial, and can be palpated under the skin; it enters the anal canal just above the subcutaneous sphincter, although the high anal fistula track runs a little deeper but still below the ano-rectal ring.

Most surgeons have favoured a simple operation. The fistula track is laid open, and converted into a flat wound by cutting away the edges of the wound. This is allowed to heal by granulation tissue; the most careful observation is necessary in the post-operative period to prevent pocketing and subsequent recurrence. The wounds take the same time as, or slightly longer than, a fissure wound to heal depending on their size.

In a consecutive series of 20 low anal or uncomplicated high anal fistulæ-in-ano the track was laid open and the flat wound so formed covered primarily with a sheet of split skin taken from the thigh or upper arm. (*Fig.* 786.) In a further case the wound was grafted on the second post-operative day (delayed primary skin-graft) because there was too much bleeding at the initial operation to warrant immediate grafting. In 16 instances the graft showed a 90–100 per cent success; in 3 cases 70–80 per cent of the graft remained viable. In 19 cases, therefore, it was possible to discharge the patient after one week in hospital with only minimal attention subsequently required. In one case, hæmorrhage under the graft

destroyed the whole of the central part; at the first dressing this dead skin was cut away and the raw area exposed was allowed to heal by secondary intention (*Fig.* 787). In a second case infection was responsible for the loss of 40 per cent of the graft.

FIG. 786.—The graft can be taken from the inner side of the left thigh. This area is left undraped in readiness.

This patient had had two previous operations for the cure of the fistula, and as a result there was considerable fibrosis which adversely influenced the blood-supply. No chemotherapy was given to this patient either pre-operatively or post-operatively. When the first dressing was done, dead skin was removed, and the wound allowed to heal by secondary intention. All patients have been followed for six to eighteen months without recurrence.

ANO-RECTAL FISTULA

The ano-rectal fistula is uncommon. Two cases were treated in this series of 23 fistulæ-in-ano. The main track may extend like a horseshoe around the anus. The opening into the bowel is nearly always below the ano-rectal ring into the anal canal. This opening cannot always be found, but unless it can be the chance of permanent cure is small (Naunton Morgan, 1949). The wound which remains after laying open the fistula is very large and the average time spent in hospital is eight weeks or longer. The longer the process of healing takes, the more indurated the wound becomes, and the more difficult it is to watch the deeper pockets and prevent recurrence.

If these fistulæ are to be treated by primary skin-grafting the surgeon must contend with the technical difficulties created by innumerable bleeding points difficult to arrest and by the large area to be grafted. Such hazards to success are overcome by adopting the principle of delayed primary closure of the wound, the most satisfactory method of treating the heavily contaminated war wound (Hughes and Webb, 1951).

Following the initial operation the wound is lightly packed with dry gauze; on the second post-operative day the patient is returned to the theatre, re-anæsthetized, the pack removed, the blood-clot irrigated away, and the wound trimmed if necessary. Sheets of split-thickness skin are cut from the thigh in sufficient quantity to cover the wound. The skin sheets are sutured to the skin edge, and to one another in the depth of the wound. The graft is then covered with tulle gras and cotton-wool is

FIG. 787.—Two separate low anal fistulæ. Both excised and treated by primary skin-graft. The upper graft was quite successful, but the central portion of the lower graft was lifted by a hæmatoma, leaving a viable peripheral ring.

firmly and carefully packed into the cavity to obtain close apposition. The wound is first dressed on the fifth post-operative day, i.e., on the seventh

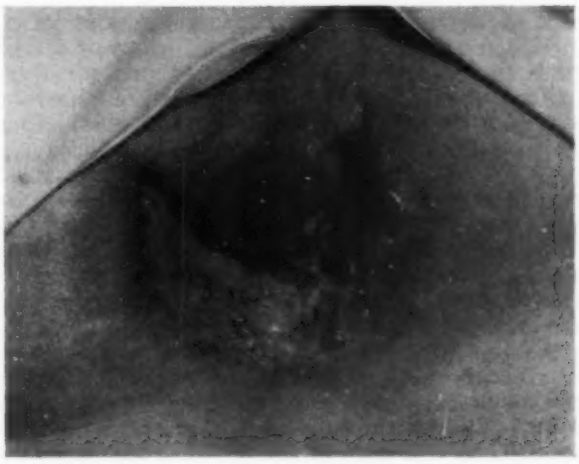

FIG. 788.—Ano-rectal fistula, treated by delayed primary skin-graft (Thiersch), photographed on the fifteenth post-operative day. Healed except for isolated small areas of granulation tissue.

day following the initial operation. The bowels are kept confined in this time.

Two patients with ano-rectal fistulæ have been treated by primary skin-grafting. The first patient, a male, had an ano-rectal fistula which at operation appeared to be confined to one side of the anal canal, and which did not appear to be connected with the anal canal. Following saucerization of the wound a split skin-graft was applied. On the first post-operative day blood was oozing from the outer edge

of the wound, indicating loss of at least part of the graft. This was confirmed when the dressings were removed on the fourth post-operative day; a large hæmatoma apparently arising from the inferior hæmorrhoidal vessels had lifted about 30–40 per cent of the total graft. The remainder of the graft was intact and healthy; however, the fistula persisted, which was not unexpected because the internal opening could not be found.

The second patient had a complete 'horseshoe' ano-rectal fistula. Following excision and saucerization the wound was packed with dry gauze. On the second post-operative day the wound was grafted. The graft was inspected five days later and a 95 per cent success was revealed. She was discharged a few days later (*Fig.* 788). The deep artificial crevices around the anus were gradually obliterated as the tissues expanded.

DISCUSSION

The importance of early skin cover of open wounds cannot be over-emphasized. The proctologist is reluctant, and rightly so, to suture the fistula wound. It is, indeed, illogical to lay open a fistula, and then suture it up again hoping that no deep hæmatoma will form and become a nidus of infection and recurrence. The procedure would be quite unsuitable for high anal and ano-rectal fistulæ.

Primary or delayed primary skin-grafting of open wounds around the anus has a sound surgical basis. The lesion, whether a fissure-in-ano, a stenosis of the anus, or a fistula-in-ano, is treated in accordance with accepted principles; the wound is covered with a split-thickness skin-graft. Nothing is lost if the graft fails in part or whole; the denuded area is left to heal by secondary intention.

There are several reasons why primary or delayed primary skin-grafting has not been adopted previously as the standard treatment in treating ano-rectal wounds. Skin-grafting wounds in this region presents technical difficulties in maintaining the graft in continuous apposition to the wound for several days. A satisfactory method for the smaller wounds which form the majority, involves the use of a mould of dampened cotton-wool or of gutta-percha. A sheet of skin cut from the inner side of the left thigh as the legs rest in the lithotomy position, is placed over the face of the mould. The mould, and skin, is placed accurately in the wound, and held firmly in position by sutures tied across the back of the mould. An alternative method is to suture the piece of skin to the edges of the wound, and obtain pressure by dressings carefully placed over the graft. Additional support to the graft can be obtained by strapping the buttocks together. The patient is kept in bed, and allowed up only after the first dressing. The graft is inspected on the fourth or fifth post-operative day, and the bowels are kept confined until this time. The first dressing can be done either in the ward or in the theatre, but it should be done with care; in one case in this series the graft was avulsed during the first dressing.

Hæmorrhage was responsible for relative failure in two instances in this series, and was the cause of minor losses of graft in others. Many of the bleeding vessels are small and are quickly stopped by firm pressure applied for a minute or two. Larger vessels will require ligature with the finest catgut. If the wound be a large one, and bleeding occurs from numerous vessels, the surgeon is advised to pack the wound with dry gauze and wait for a day or two before applying the graft; delayed primary grafting is likely to be successful where an immediate primary graft would not.

The ano-rectal region is heavily contaminated and wound infection has always worried the proctologist who tries for wound healing by first intention. In this series a pre-operative course of intestinal sulphonamide has been given, and, following operation, penicillin and sometimes streptomycin. It is difficult to estimate the importance or otherwise of the chemotherapeutic and antibiotic cover. In one case it was not used and infection destroyed part of the graft, but there were other factors which might have been responsible, for example, the poor bed for the graft consequent upon two previous operations. With gentle and careful operative technique and complete control of hæmorrhage, chemotherapy is probably unnecessary.

SUMMARY

1. Ano-rectal wounds have been treated cautiously in the past and many surgeons still prefer to leave the wounds open and allow healing to proceed by the slow but relatively safe 'secondary intention'. Few proctologists favour suture of these wounds, and some wounds, such as those which follow treatment of an ano-rectal fistula, cannot be so treated. The most satisfactory method involves the application of a split-thickness (Thiersch) graft to the wound either primarily, or two or three days later as a delayed primary graft.

2. This method of treating wounds is safe, reliable in most cases, and considerably shortens hospitalization and the convalescent period.

3. Practice overcomes the technical difficulties associated with placing and keeping a graft in position in this area. Hæmorrhage can be controlled by pressure and by fine ligatures, but if hæmostasis be imperfect the surgeon may wait for a day or two and allow the smaller vessels to close spontaneously and then perform a delayed primary skin-graft before any granulation tissue has appeared.

4. The results of primary and delayed primary skin-grafting in a series of cases suffering from fissure-in-ano, anal stenosis, and fistula-in-ano are reported.

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RECTAL BLEEDING AND DIVERTICULITIS

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"In a middle aged or elderly patient apparently in fair health, has a sudden, unexpected, and alarming profuse rectal hæmorrhage of perhaps several pints, full investigation is likely to reveal diverticulosis of the colon and the bleeding is unlikely to continue."

I first heard this proposition put forward by Mr. D. C. Corry. Few of my colleagues appear to recognize this association and consultation of the more popular text-books (Price, 1950; Osler and McCrae, 1926; Savill (Warner, 1950); Bailey and Love, 1948; Aird, 1949; Handfield-Jones and Porritt, 1951; Romanis and Mitchiner, 1948; Pannett, 1947; Lockhart-Mummery, 1934) reveals no reference to it. When mentioned at all, such bleeding is attributed to carcinoma. However, several cases encountered confirmed the statement and the subject, therefore, appeared worthy of further attention. This statement is of great clinical interest, but even if it is accepted as true every patient must be most thoroughly investigated.

It is our experience, although our figures are insufficient to prove this particular point, that in a case of diverticulitis the bleeding will stop and that a week or more later three separate stools will be clear of occult blood. On the other hand, in cases of carcinoma of the colon we have not seen three separate stools all give a negative test for occult blood. A single stool is never to be relied upon.

Here are two typical histories to illustrate the syndrome:

E. B. (R.I. 130248), aged 74, was perfectly well until Sept. 2, 1950. There had been no pain, no change in bowel habit, no blood or slime in the stools. In the evening he had an unexpected call to stool, and passed about a pint of blood. During the next one and a half hours he passed five such motions, each succeeding one containing less blood, the last one about half a pint. There was no pain throughout. Clinical examination showed no abnormality, a barium enema demonstrated extensive diverticula, and three sigmoidoscopic examinations revealed no lesion. There were no further symptoms during a six-months' follow-up.

T. W. (R.I. 8033/43), aged 72, had been quite well apart from arthritis of the hip-joints and had had no symptoms of any type referable to his digestive tract. On May 19, 1952 he woke up at 2 a.m. thinking he had inadvertently passed a stool in bed. On investigating he found he had passed bright red liquid blood. He then passed a further 6 or 7 motions of pure blood, estimating his loss as 2 pints. On examination no abnormality was found apart from a blood-pressure of 200/115 mm. Hg. His hæmoglobin fell to 54 per cent after compensation and he was transfused. Subsequent investigation by barium enema revealed only diverticulosis. He has had no further trouble.

THE LITERATURE

The volume of writings on diverticulitis is now so considerable that only an incomplete review is presented; references to a few cases of the type under discussion appear scattered throughout it. Stone (1946) says: "It should be borne in mind that the outstanding symptom (of diverticulitis) may be sudden profuse bleeding into the bowel." Previously he had included in a paper on "melæna of obscure origin" (Stone, 1944) 8 cases similar to those which we are considering, all except one over 60 years old. Pfeiffer (1944), in the discussion of this paper, had confirmed this statement.

Bacon (1949) found bleeding in some form in 17 out of 107 personal cases and Morton (1946) found "bright blood" in 24 of 111 cases, "a much higher figure than expected".

Rosser (1939) states: "Bleeding has been a frequent concomitant or presenting symptom in individuals with diverticulitis." Of 24 consecutive cases of diverticulitis 6 had bled, and in 16 patients investigated for rectal bleeding no cause beyond diverticulitis was found in 8, 4 of which had had alarming hæmorrhage. In all cases the bleeding ceased spontaneously. Mayo and Blunt (1950), analysing 202 cases of diverticulitis treated by resection, include 16 complaining of melæna and 1 with a severe rectal hæmorrhage; but the type of case we are discussing rarely requires resection.

Germain, Le Gallou, and Morvan (1938) describe admirably one case of the syndrome under discussion as a new variant of diverticulosis.

Boyden (1950) found hæmorrhage the presenting sign in 3 out of 34 cases of diverticulitis treated surgically, and Cave (1942) stated: "It is the experience of most of us that patients with the history of rectal bleeding, sent to the roentgenologist for a barium enema, return with a report of diverticulosis or diverticulitis and not cancer."

On the other hand Harold Edwards (1939) in his admirable book mentions only one case of severe bleeding with diverticulitis, and says that bleeding should not be regarded as a characteristic of the condition. Rankin and Graham (1939) state: "Blood in the stool is of little significance in diverticulitis, from a diagnostic standpoint, and although it is present in a number of cases, usually proctoscopic examination will reveal that the presence of the diverticulitis had little, if anything, to do with the presence of the blood." Similarly Eggers (1931) in 24 cases of diverticulitis found bleeding in 6, but said that it was not an important symptom and if present should make one think of a complication or of a carcinoma. Later he (Eggers, 1941) reported bleeding in 16 cases out of 82. David (1946), discussing Stone's (1946) statement, disagrees: "It is always possible that small adenomas or nevi may be the source of relatively minor hemorrhage and it has been well-pointed out that large or massive hemorrhage may come from sources undiscoverable even at autopsy."

This last statement apart, it will be clear that some of the contradictions in the literature are due to a failure to distinguish between the sudden, massive, alarming hæmorrhage without preceding symptoms which we are considering, and the frequent presence of small quantities of obvious or occult

blood with which we are not now concerned. Such blood is, in fact, remarkable by its constant absence in these cases.

CASE RECORDS

To get some further light on the syndrome an attempt was made to form an impression of its incidence from a study of the records of patients admitted to the Radcliffe Infirmary in the years 1946–51. Case notes indexed by the final diagnosis and not by the leading signs and symptoms presented an immediate difficulty. All available relevant histories which were sufficiently complete were, therefore, submitted to close personal investigation. There were 46 cases coded as ' diverticulosis/diverticulitis ', 39 of ' rectal bleeding ', 109 of ' melæna ', 76 of ' hæmorrhoids ', and 96 of ' neoplasm of colon and rectum ', a total of 366.

Of the 46 cases of diverticulosis diagnosed by barium enema and sigmoidoscopy 10 had had severe bleeding. Their ages were 53, 59, 60, 62, 63, 65, 70, 70, 71, and 74 years. Their blood-pressures were 195/115, 175/105, 140/70, 120/80, 134/80, two not recorded, 135/75, 140/85, 150/100 mm. Hg, a fair cross-section for this age group. The bleeding was estimated from ½ to 3 pints, it occurred once only in 6 cases, three times in 2, and on six successive days in 1 case. There has been no further bleeding in any of them. In 1 case the bleeding followed a pre-operative enema, in 4 cases there was some colicky lower abdominal pain preceding the passage of blood, in 2 some diarrhœa, with vomiting in 1 case. The barium enema showed very extensive diverticulosis in all these cases.

Of the 39 cases of ' rectal bleeding ' 5 presented the syndrome. They were aged 51, 59, 65, 72, and 79 years. Their blood-pressures were 110/65, 170/100, 200/115, 130/75, 120/85 mm. Hg, and the bleeding occurred once only, estimated at ½ to 3 pints. Two of them were transfused and there was slight further bleeding in 1 case. In 1 case the bleeding was preceded by colic and diarrhœa. Again full investigation revealed only very marked diverticulosis in these cases.

Of the 109 records classified as ' melæna ' and the 76 of ' hæmorrhoids ' none have any resemblance to our syndrome.

In the 96 cases of ' neoplasm of colon and rectum ' 1 case presenting somewhat similar symptoms was encountered, and since this is unusual and might be considered to vitiate the whole thesis of this paper, his history is given briefly :

W. R. (R.I. 34699/43), a doctor aged 80, began to have attacks of constipation and colicky pain four years before admission. They were ' cured by aperients ' and were of irregular incidence. Four months before admission he said he had a ' sudden hæmorrhage of several pints ' by the rectum while in bed at night, and occasional slight bleeding thereafter. He then began to have symptoms and signs of obstruction, and on admission was found to have an annular carcinoma of the splenic flexure of the colon.

This history differs from those of the diverticulitis cases in that there were definite abdominal symptoms before the hæmorrhage occurred and the bleeding continued.

DISCUSSION

It would seem reasonable to conjecture that the bleeding arises from erosion, by infection or concretions, of the vessels which, in penetrating the muscular wall of the bowel, give rise to the weak spots through which diverticula are protruded. This would account for the severity of the bleeding but does not explain the fact that the bleeding seldom recurs. Mr. Corry saw a private patient eight years ago (not included in this series) who had had a sudden hæmorrhage of 5 pints in the typical manner. He was investigated and diagnosed as diverticulitis. He kept quite well until last year, when he bled again profusely. There were no new findings on investigation.

The reader will have noticed that some difficulty has been encountered in deciding whether to regard the underlying lesion as diverticulosis or diverticulitis. Often there are no other clinical manifestations of diverticulitis, and yet diverticulosis is defined by most authorities as symptomless. Further, it is difficult to explain the bleeding without postulating some inflammatory change however slight and however narrowly confined. It therefore seems safer to refer to the condition as diverticulitis, although many of the reports on the barium enemata mention ' very marked diverticulosis'.

In conclusion it is felt that however unscientific and statistically unsound this brief survey may be, it does indicate the usefulness of the introductory statement as a rough clinical signpost. When an elderly person suddenly has this type of bleeding there is likely to be profound alarm and despondency, yet it should be noted that none of these patients died. The treatment in all cases was, of course, conservative, with blood transfusion when required. It is worth while repeating, however, that the fullest possible investigation is essential in all cases as soon as the immediate crisis is past, and that a careful follow-up is necessary, with repeated stool examinations for occult blood on three separate specimens, sigmoidoscopic examinations, and barium enemata.

SUMMARY

The relationship, in the elderly, of heavy, sudden rectal bleeding without warning symptoms to diverticulitis is examined. Brief reference to the literature is made. The study of 366 case histories indicates that such a relationship does exist.

It is a pleasure to thank Mr. D. C. Corry for allowing me to elaborate one of his teachings and for constructive criticism ; the Physicians and Surgeons of the Radcliffe Infirmary for access to their case notes ; and Miss B. Merry, of the Records Department for much time and trouble in collecting these.

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MEIGS' SYNDROME

A BRIEF REVIEW OF THE CONDITION AND REPORT OF A CASE ASSOCIATED WITH A BRENNER TUMOUR

BY B. C. ROWLANDS, J. V. GARRETT, AND P. A. KING

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ASCITES and hydrothorax associated with fibroma of the ovary was first described as a separate clinical entity by Meigs and Cass (1937), and its occurrence is now generally recognized. The awareness of such a condition is important if a diagnosis of inoperable malignancy is to be avoided in those cases where the primary condition is an innocent one. The case presented here is unusual in that the syndrome was associated with a Brenner tumour.

CASE REPORT

Mrs. L., aged 56, complained that for the preceding year she had noticed progressive swelling of her abdomen. For six months she had become progressively more breathless on exertion and had had occasional attacks of mild lower abdominal pain.

ON EXAMINATION.—The patient was an obese woman with orthopnœa and a brassy cough.

Circulatory System: Pulse 86, regular. B.P. 180/110. The heart appeared normal. There was no evidence of œdema over the sacrum or of the ankles.

Respiratory System: There was clinical and radiological evidence of a right-sided basal pleural effusion (*Fig.* 793).

Abdomen: The abdomen was obviously swollen and the umbilicus everted. A large mobile mass was palpable which seemed to arise in the pelvis and extend above the level of the umbilicus. The surface was hard, irregular, and easily palpable through the everted skin of the umbilicus. There was clinical evidence of ascites.

INVESTIGATIONS.—

Urine: N.A.D.

Blood: Hb 80 per cent; R.B.C. 4,500,000/c.mm.

Serum-proteins: 7·4 g./100 ml.
Serum-albumin 4·7 g./100 ml.
Serum-globulin 2·7 g./100 ml.

Radiography of chest: The findings confirmed a right-sided pleural effusion.

AT OPERATION (Feb. 6, 1953, Professor R. St. Leger Brockman).—The abdomen was opened through a lower right paramedian incision. The peritoneum contained three pints of clear, straw-coloured fluid which was aspirated. A large, right-sided, freely mobile, solid ovarian tumour was found and removed with the Fallopian tube (*Figs.* 789, 790).

PROGRESS.—The patient made an uninterrupted recovery from the operation and was discharged from hospital on the fourteenth post-operative day. A radiograph of the chest five weeks from the day of operation shows the effusion to have been almost completely absorbed (*Fig.* 794).

PATHOLOGY.—The specimen received in the laboratory was a roughly wedge-shaped, hard, white neoplasm, with an irregular lobulated surface. It weighed 990 g. and measured 16·5 × 16·5 × 12·5 cm. The Fallopian tube was stretched over one side of it and a small piece of normal ovarian tissue could be recognized. The cut surface of the neoplasms showed a fibrous whorled appearance, except for a smooth-walled, empty, cystic cavity 6 cm. × 4 cm.

The histological picture is typical of a Brenner tumour of the ovary. Epithelial cell nests are seen lying in a fibrous stroma (*Fig.* 792). Some of these nests have a pseudocystic cavity in the centre and contain a structure resembling a cumulus cöphorus (*Fig.* 791). Others have a cavity lined by mucous-secreting cells and contain mucin. The wall of the large cyst in the tumour is lined by a low columnar type of epithelium.

DISCUSSION

The earliest report of innocent ovarian tumour associated with ascites and hydrothorax was made by Cullingworth (1879). Lawson Tait (1892), in a paper read before the Royal Medical and Chirurgical Society of London, drew attention to "The Occurrence of Pleural Effusion in association with Disease of the Abdomen". He described 2 cases of ovarian tumour with ascites and hydrothorax, one malignant and the other innocent. This latter case presented with an abdominal tumour, ascites, and bilateral

pleural effusion. The diagnosis of advanced malignant disease was made, so no operation was done. During the following year the abdomen was tapped over thirty times, and as the patient's general condition remained good operation was performed fifteen months after the original diagnosis; a large ovarian fibroma was removed. The patient recovered and the ascites did not recur.

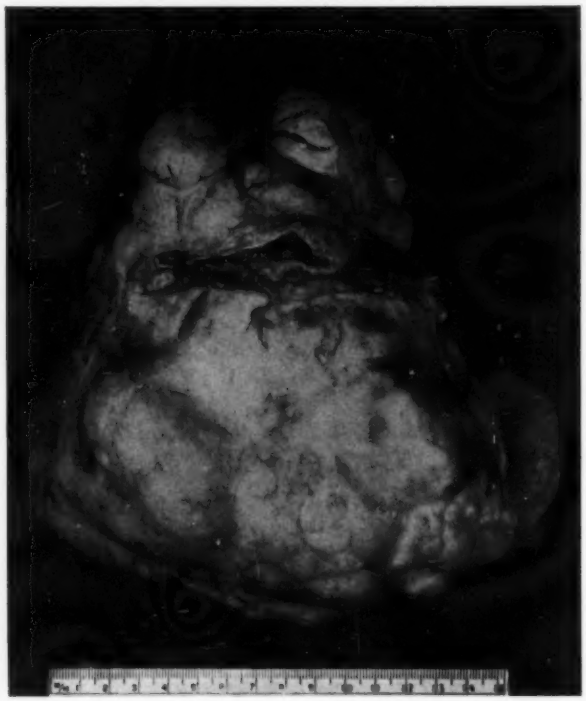

FIG. 789.—Posterior view of tumour, showing Fallopian tube stretched across it.

FIG. 790.—Cut surface of tumour, showing whorled structure and cystic spaces.

Following these early cases, other occasional cases are reported in the literature. Since the paper by Meigs and Cass (1937), however, the syndrome has been recognized as a definite clinical entity. These authors only mentioned ovarian fibromata in their series, but now it is known that other benign tumours produce the syndrome. Other writers have also included cases of papillary cystadenocarcinoma and malignant cystadenoma, in which the associated ascites and hydrothorax have disappeared following operative removal. Including all such cases, Sardi and Guixa (1949) collected 74 cases from the literature, to which they added 1 case of their own. Of the 75 cases, 62 (72·2 per cent) were solid tumours, usually fibromas, 5 were cystadenomas, and 8 cystadenocarcinomas. One other case associated with a Brenner tumour has been reported by Kelemen (1944). Fox (1942), in a review of 166 cases of Brenner tumours in the literature, does not mention the occurrence of hydrothorax and ascites in any.

The rareness of the condition may be judged from the figures of Calmenson, Dockerty, and Bianco (1947), who could find only 9 cases of Meigs' syndrome in 20,000 records of pelvic tumours, removed surgically, at the Mayo Clinic between 1910 and 1945. Sardi and Guixa's case was the only case of Meigs' syndrome occurring in 405 cases of ovarian tumours observed between 1931 and 1947 in a large South American Clinic.

The clinical findings in the case reported here conform very closely to those found in the series of cases recorded by Calmenson and others (1947). These authors found the most common complaint was swelling of the abdomen; shortness of breath was the next, and some patients had a dry cough. Lower abdominal pain, when it occurred, was mild in degree. Physical examination reveals in most cases a pelvic tumour, associated with ascites and hydrothorax. Sometimes, however, the ovarian tumour may be so small that it is only detected after paracentesis. The pleural effusion may only be diagnosed after a routine X-ray examination of the chest.

A malignant ovarian tumour with ascites and thoracic metastases is the condition most likely to be confused with Meigs' syndrome, a mistake so well illustrated in the case described by Lawson Tait. The clinical picture may also be mistaken for an advanced neoplasm of the intestinal tract with peritoneal metastases and ascites, and, if this condition is suspected, it may also be wrongly assumed that if a hard mobile pelvic mass is present it is a Krukenberg tumour. The differential diagnosis from medical diseases has been comprehensively discussed by Crosnier and Mougenot (1950). They emphasize that cardiac, renal, and hepatic causes of effusion must be carefully excluded, and initial investigations should be medical rather than gynæcological. They also point out the value of diagnostic paracentesis for the exclusion of tuberculosis.

Aetiology.—It is only natural that in the absence of an unequivocal explanation of the syndrome, there should be a wide divergence of opinion regarding its production.

Certain features of the condition are significant but do not themselves lead to any constructive conclusion. It is known that the plasma-protein estimations are usually within normal limits, though some depletion may ensue if abdominal paracentesis is repeated often. Alteration in the albumin-globulin ratio has never been noted in the absence of a coexisting medical condition capable of producing it. In many cases it has been noted that following the operation the hydrothorax has been rapidly absorbed and does not recur (Conway-Hughes, 1951). In the present case a very substantial reduction had occurred within twelve days of operation. The hydrothorax, as in this case, is commonly right-sided. Of the 10 cases recorded by Calmenson and others (1947), 8 were right-sided.

Meigs (1939), discussing the aetiology, appears to favour Selye's alarm reaction (Selye, 1936). This reaction is the immediate response of the body to an irritant or noxious stimulus, such as cold or injury.

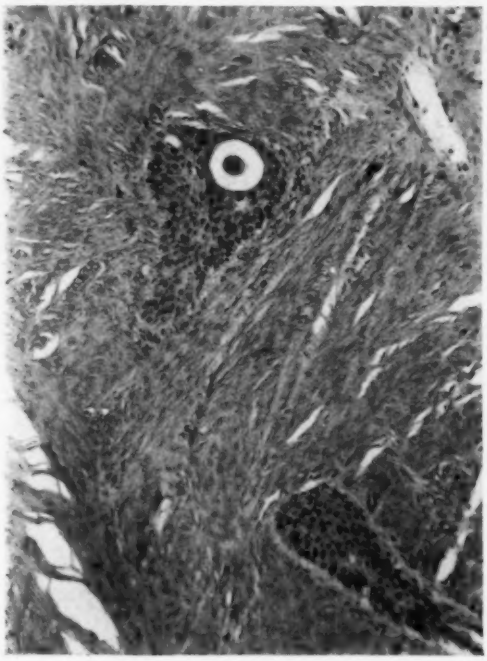

FIG. 791.—Photomicrograph showing structure resembling cumulus oöphorus. (× 88.)

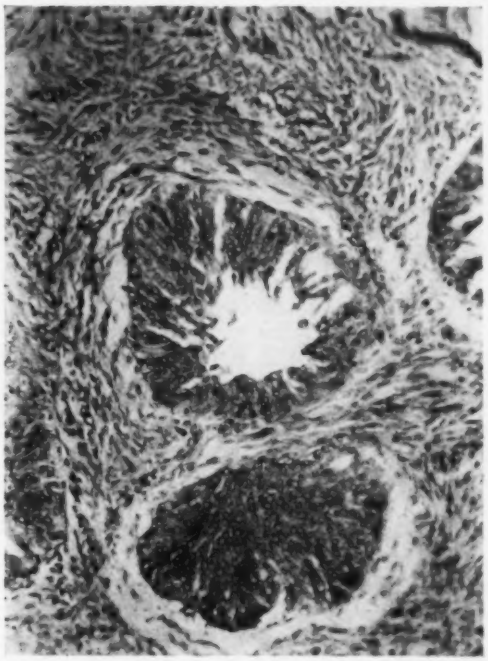

FIG. 792.—Photomicrograph showing epithelial islands in fibrous stroma. (× 88.)

FIG. 793.—Pre-operative radiograph of chest demonstrating right-sided pleural effusion.

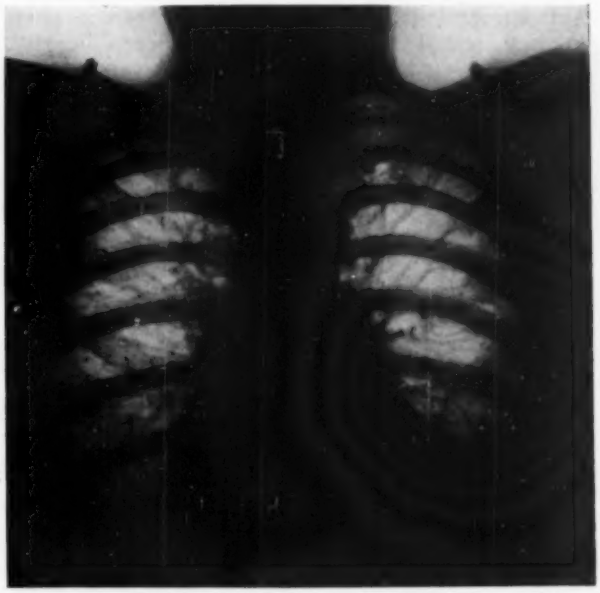

FIG. 794.—Radiograph of chest five weeks following operation. The pleural effusion has completely absorbed.

It is brought about by the release of adrenal cortical hormones, and in animals may include the production of ascites and hydrothorax. It seems unlikely, however, that this immediate response to a stimulus of limited duration can be reasonably considered as the cause of a condition associated with a chronic illness. Calmenson and others (1947) also do not consider the alarm reaction hypothesis a satisfactory explanation of the syndrome. Another point is the fact that the pleural effusion is usually unilateral, whereas, if it were part of a general response to a noxious stimulus it would more likely be bilateral.

One explanation to account for the ascites is that the tumour, by virtue of its weight and size, may compress sufficient large abdominal lymph-vessels to cause a transudate. This, however, still leaves the pleural effusion to be accounted for, and, in any case, is not a satisfactory explanation of those cases in which the syndrome is associated with a small ovarian tumour incapable of occluding lymphatics. It also does not account for the non-appearance of ascites in pregnancy and other large pelvic masses. The theory put forward by Rubin, Novak, and Squire (1944) and Calmenson and others (1947) that the ovarian tumour is œdematous and the œdema fluid permeates its capsule, so causing the ascites, also does not seem very likely. The tumour from the case described here was not œdematous.

The possibility of patent pleuro-peritoneal canals has been suggested to account for the coexistence of ascites and hydrothorax. If this were the case then paracentesis should also drain the thoracic effusion, but it does not. Ritvo (1942) introduced air into the peritoneal cavity of a patient with Meigs' syndrome and immediate radiography of the chest did not reveal any air in the pleural cavity. In general, it is unlikely that the syndrome will be explained by any existing anatomical anomaly.

Finally, the possibility that the ascitic fluid is transferred to the pleural cavities via the trans-diaphragmatic lymphatics must be considered. There is definite experimental evidence that particulate matter introduced into the peritoneal cavity can be transferred by lymphatic channels into the pleura, and Meigs demonstrated such transference in two cases of his syndrome. Burger (1949) favours this theory, but admits that it fails to explain the particular predeliction for a right-sided hydrothorax and the absence of hydrothorax in cases of ascites of other origin. It seems, however, to be the best explanation so far put forward.

SUMMARY

A case of Meigs' syndrome associated with a Brenner tumour of the ovary is presented and theories as to its mode of formation are discussed. We would enter a plea for exploration of any case suggesting the syndrome.

Our thanks are due to Professor R. St. Leger Brockman and to Dr. H. P. Brody for permission to publish this case. The illustrations are the work of Mr. Brook, of the Photographic Department.

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A LARGE RETROPERITONEAL FIBROMA

BY SURGEON COMMANDER DEREK STEELE PERKINS, ROYAL NAVY

THE incidental discovery of an almost symptomless, easily palpable, abdominal tumour is stimulating to the clinician and provides discussion amongst one's colleagues. This case is unusual in that this man presented with a tumour approximately 6 in. in diameter, in the lower half of his abdomen—of which he was quite unaware.

CASE REPORT

The patient, a male officer aged 32, was seen in a Royal Naval Hospital during the Autumn of 1952. He had been sent in by the Medical Officer of his ship for a medical examination because he was depressed, introspective, and his only objective symptom appeared to be a slight swelling of one or other testicle from time to time.

He was a well-built man, rather overweight, but otherwise appeared quite fit. His history was that as a child he had been backward and had not walked until the age of 7. During adolescence he held his own and enjoyed normal activities to the full, and at the age of 20 obtained a Commission in the Royal Navy. A few years later he married and has one healthy child.

In 1949, for no apparent reason, he had hæmaturia on one solitary occasion—this was investigated and no cause was found. The symptom did not recur. About the end of 1951 he became aware of neuralgic pain in his left testicle and thought that at times it was swollen—then the same symptoms occurred in the right side, and then alternated. He worried about this, but was reassured and began to wonder if, in fact, his complaints were real or imagined.

About mid-June 1952 he joined a Mess where living was excellent and his duties were mainly sedentary. Then he noticed his abdomen was increasing in size, but concluded that this was only to be expected in the foregoing circumstances, especially as in his new surroundings

FIG. 795.—The small gut is displaced upwards to the level of the pelvic brim, showing a smooth outline with the concavity downwards.

he enjoyed duty-free liquor. However, his new appointment gave him much time to think about his own troubles and this culminated in his examination in hospital.

ON EXAMINATION.—Between the symphysis and the umbilicus was a smooth firm swelling lying slightly to the

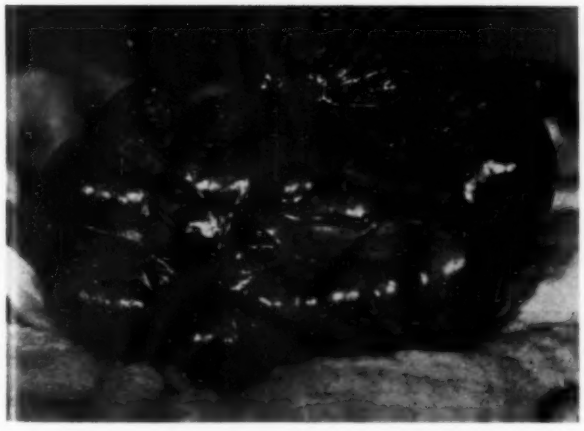

FIG. 797.—The tumour in situ before removal.

right of the midline—about the size of a large grapefruit. It could be moved freely a limited amount in all radial directions and felt as though it were anchored posteriorly. It did not move on respiration. Rectal and sigmoidoscopic investigations were negative. Both testicles and cords felt normal.

Investigations.—Normal blood-picture ; negative W.R.

Barium meal follow-through showed no hold-up, but in the 1-hr. and 2-hr. films, and on screening at 2½ hr., it was noted that the small gut was displaced upward to the level of the pelvic brim, showing a smooth outline with the concavity downwards (*Fig.* 795).

FIG. 796.—Rather tortuous ureters and a fullness of proximal half of both ureters and renal pelves.

Intravenous pyelography showed somewhat tortuous ureters with a rather unusual fullness of the proximal half of both ureters and renal pelves (*Fig.* 796).

The urine contained a few red blood-corpuscles, many leucocytes, and a light cloud of albumin.

FIG. 798.—Section of tumour on removal.

AT OPERATION.—A right oblique muscle-cutting incision—peritoneum opened and a tense lobulated purply-red mass presented (*Fig.* 797)—this was found to be attached by quite a small, very vascular pedicle to the posterior abdominal wall in the region of the promontory ; it was removed by isolating groups of vessels and ligating them, and oversewing the base with a peritoneal flap.

The tumour weighed 2 lb. and was retroperitoneal. The contents of the abdomen were examined and appeared to be normal. The wound was closed in layers without drainage. Recovery was uneventful.

Histopathology.—A retroperitoneal fibroma. "Microscopically this tumour, measuring 14 × 13 × 11 cm., is blotchy, reddish-purple in colour, lobulated and well supplied with blood-vessels. All but an area of 7 × 7 cm. is covered by peritoneum, which is smooth and intact.

FIG. 799.—Showing the structure of the tumour. (× 60.)

The bare area is covered by loose areolar tissue, under which the tumour has a lobulated surface, but does not appear to have invaded or penetrated any adjacent tissue. The cut surface of the tumour is whitish, firm, and fibrous." (*Fig.* 798.)

Professor G. Hadfield, of the Royal College of Surgeons, kindly prepared the photomicrograph and made the following histological report: "The tumour has the structure of a fibroma with some modification due to œdema and lymphatic dilatation." (*Fig.* 799.)

DISCUSSION

Simple retroperitoneal tumours are rare (Goebell, 1901). A search through the literature concerning the frequency of these tumours confirms this view, and of those recorded only a proportion have been confirmed by histopathological examination (Wolfer, 1934).

The opinion held by many is that most retroperitoneal tumours are sarcomata unless growing from, or involving, the kidney. The fact that patients with these tumours (retroperitoneal) may lose weight rapidly, synchronizing with the rapid growth of the tumour, and its hardness (Ogilvie, 1930) on palpation, further add to this conception. In discussing the diagnosis of retroperitoneal tumours those obviously coming from the pancreas, kidney, adrenals, or retroperitoneal lymph-glands, primary or secondary, will not come under discussion. Of the other tumours, however, fibromyoma, neuroblastoma, teratoma, and cysts, because of the similarity of the clinical signs and symptoms, must be considered (Wolfer, 1934).

These signs depend on the location of the tumour—whether it is large or small and whether it is growing rapidly. In a large percentage of cases the only early symptom is a vague sense of weight and discomfort in the abdomen, and may extend over years. Dysuria, displacement of the bladder, and dilatation of a ureter have been recorded (Walters, 1935). Pain usually appears later and may be referred to the testicle or down the leg. Œdema of penis and scrotum has been reported as a symptom and as the tumour increases in size, usually it becomes more fixed (Muller, 1936). The tumour may imprison and drag other organs with it, involving the aorta, and makes removal difficult or impossible (Cope, 1927).

SUMMARY

A case of a simple retroperitoneal fibroma, weighing 2 lb. In its early stages of development the symptoms were slight but clinical examination at once revealed its presence. The clinical features of these tumours are not characteristic, but a palpable mass is invariably present by the time the tumour starts to produce symptoms (Herdman, 1953).

Radiography is usually of great value, especially urography and barium follow-through. The treatment is excision.

The writer would like to thank Sir Cecil Wakeley (Surgical Consultant to the Royal Navy) for his help in this case; Professor G. Hadfield, for the photomicrograph and histological report; and to the Medical Director-General of the Royal Navy, for permission to publish this case.

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THE STOMACH AS A CONTENT OF INGUINAL AND FEMORAL HERNIÆ

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THE stomach is a rare occupant of the sac of an inguinal hernia and is yet more rarely found in a femoral hernia. The discovery of the stomach in the inguinal hernia of a patient of ours led to a prolonged but fascinating search of world literature in an effort to trace all the recorded cases. Towards the end of this search one of us was asked to see a patient with a very large femoral hernia and a barium meal confirmed our suspicions that the stomach might be in the sac. We are able, therefore, to report two new cases—one of stomach in an inguinal hernia and one of stomach in a femoral hernia, together with a summary of the cases already published.

STOMACH IN INGUINAL HERNIA

CASE REPORT

HISTORY.—E. A., a man of 61, was sent into hospital by his doctor as a case of strangulated left inguinal hernia. He was a house painter and had had a left inguinal hernia for about forty years. He had worn a truss intermittently. There was no history of dyspepsia nor had he noticed any alteration in the size of his large hernia after eating or drinking. The hernia had always been reducible up to the day before admission to hospital.

He had only worked for one month during the past year because of bronchitis and he had been in bed for the last two months with dyspnœa and œdema of the feet. Six weeks previously he had a right-sided pulmonary embolus.

Two days before admission he vomited dark fluid on about ten occasions but had a normal bowel action. The day before admission he vomited continuously and was not able to reduce his hernia, which became painful. There was no abdominal pain.

ON EXAMINATION.—A frail looking man, who was pale, cyanosed, very dyspnœic, and constantly vomiting. No œdema of legs.

Abdomen: Markedly distended and tense. Very large left scrotal hernia, which was neither tense nor tender and varied in size as he strained to vomit. The hernia could not be reduced.

Chest: Very poor expansion, with scattered rhonchi and basal râles.

Cardiovascular System: The pulse was of poor volume. Rhythm regular and apex beat in the anterior axillary line. B.P. 120/80.

TREATMENT.—A Ryle's tube was passed and aspirated 137 oz. (3·89 l.) of dark fluid, smelling of bile, and some of which had the 'coffee grounds' appearance. The aspiration produced a dramatic improvement, the abdomen being greatly reduced in size and quite lax. The hernia could be reduced and the left external ring was found to admit three fingers and the right two. He was much less dyspnœic and the pulse volume improved.

He was put on an intravenous drip and gastric suction till the fourth day, when the aspirations were of such quantity and quality that oral feeding was commenced. A straight radiograph of the abdomen had not shown any fluid levels. Three days later, as he was feeling nauseated, a tube was passed and a litre of brown fluid aspirated from the stomach. It was then thought that he had pyloric stenosis rather than a mechanical obstruction, and the hernia appeared to be unrelated to the symptoms for which he was admitted. However, the following was reported after a barium meal: "The barium-filled stomach is herniated into the scrotum, likewise small intestine. There is a moderate four-hour gastric residue. No ulcer seen in the stomach, and the duodenum was not demonstrated (*Fig.* 800). At 24 hours the transverse colon occupied the sac and the stomach was empty."

The patient's condition steadily improved and he was able to take a normal diet. It was noted while standing

FIG. 800.—Radiograph of barium meal showing part of the stomach in a left inguinal hernia.

that his hernia reached to a point 11 in. (28 cm.) below the great trochanter and that his height was only 62 in. (1·57 m.). When it was found that he could sleep without distress with his hernia reduced, he was considered fit for repair of his hernia, four weeks after admission.

AT OPERATION (W. W. D.).—Local anæsthesia 1–1000 amethocaine hydrochloride. The sac was opened and found to contain omentum, small intestine, pelvic colon, and the body of the stomach (*Fig.* 801). It was an indirect hernia and there were dense omental adhesions and a sliding hernia of the colon on the medial wall. Some omentum and the testis were removed and the remaining contents reduced without difficulty. The neck of the sac, which admitted four fingers, was closed with a purse-string of Chinese twist silk and the sac cut across. The conjoined tendon was sutured to the inguinal ligament with interrupted silk sutures after a Tanner relaxation incision had been made.

RECOVERY.—This was uneventful. A right inguinal hernia became apparent before he was discharged. He was fitted with a truss as he did not desire another operation.

A barium meal six months later showed the stomach and duodenum in their normal position (*Fig.* 802). His left inguinal hernia was soundly repaired and the right inguinal hernia satisfactorily controlled by a truss.

REVIEW OF CASES ALREADY PUBLISHED

Of the 34 cases already recorded of stomach in inguinal hernia, only 5 are in the English language. No previous article in English has given a list of the published cases and we therefore give in *Table I* a summary of these cases, which has been compiled from the original articles.

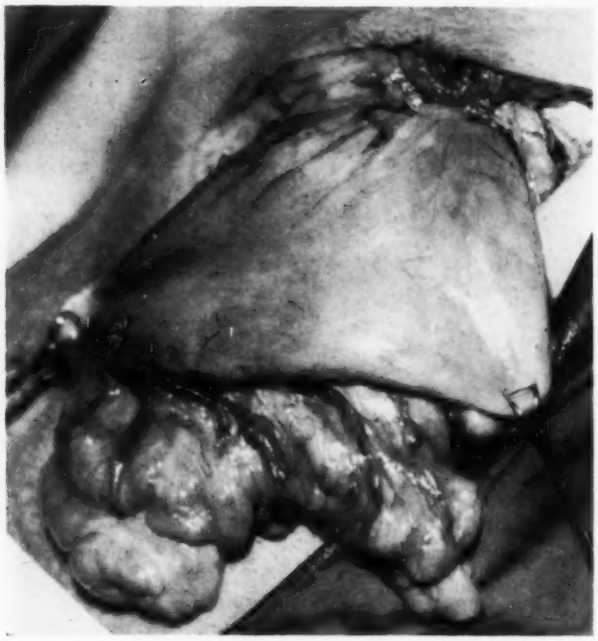

FIG. 801.—Photograph of stomach in left inguinal hernia exposed at operation (case shown in *Fig.* 800).

FIG. 802.—Radiograph of barium meal six months after operation, showing stomach in normal position (case shown in *Figs.* 800 and 801).

The first case was published in 1802 by " le citoyen Lallement, professeur de l'Ecole de Médecine de Paris ". It is of special interest not only because it is the earliest we have found, but also because of the detailed medical history given of the patient, Pierre Persain, from the appearance of the hernia at the age of 32 until his death 32 years later. Pierre suffered much abdominal discomfort during this long period and autopsy disclosed the pyloric portion of the stomach in his right inguinal hernia. Lallement states that when eating, Pierre felt the food fall into the hernia, which became bigger, heavier, and more painful to the point that following a meal he was obliged to unbutton himself and support it with his hands. During the two hours following a meal, he felt food being carried with gurglings from the hernia to the epigastrium and stay there for some time. Although he makes no claim, Lallement must have suspected the diagnosis while Pierre was alive.

The third case forms one of the subjects of a thesis by Fabre (1832) for the M.D. degree of Paris. It is of special interest because a clinical diagnosis of stomach in a right inguinal hernia was made by Fabre during the lifetime of the patient and confirmed later at autopsy by Lebert (1838). Unlike Lallement's patient, this one had no digestive symptoms, but percussion of the bilateral herniæ before and after food led Fabre to conclude that a portion of the stomach was in the right inguinal hernia. Further confirmation was obtained by noting the increased size on the right after taking food and feeling with the hand the impact of the fluid entering the stomach in the scrotum while the patient was drinking. Fabre was most anxious that when death occurred, the medical attendant should have the opportunity of confirming his confident diagnosis and, as already mentioned, Lebert was able to do this. We do well to salute the memory of M. Fabre.

With the exception of Fabre, the diagnosis in all the early cases was first made at autopsy. Some of these patients had acute symptoms due to obstruction of the stomach by the hernial ring and a diagnosis of strangulation or incarceration was not unnaturally made, although the classical signs of strangulation were not usually present. Brunner (1897) was the first person to record a case where an emergency operation for strangulated hernia had disclosed the stomach in the hernia. Brunner's patient made a good recovery, but, without wishing to detract from a remarkable achievement, it may be noted that his patient was only 28 and had a medium-sized hernia, while the majority of such patients have been over 60 and have had large herniæ.

Rieder (1915) was not only the first person to diagnose the presence of the stomach in a scrotal hernia by radiography, but with periodic opaque meals he watched the gradual descent of the stomach into a left inguinal hernia over the course of three and a half years. After having made the diagnosis in this way, he was able to hear and feel the descent of fluid

into the hernia and noted its enlargement after drinking. Elischer (1923) is the only person who has recorded two cases of stomach in inguinal hernia, and he carried out successful emergency operations on them both. Two cases have been recorded in women, and after diagnosing the presence of the stomach in a woman's left inguinal hernia by radiographs, de Vernejoul and de Luna (1925) were the first to report a case treated by elective surgery. Sicot (1927) and Oakley (1937) described patients on whom emergency surgery was undertaken for strangulated inguinal hernia, and in each case a portion of the stomach with a perforated gastric ulcer was found in the sac.

The case we describe appears to be the first recorded in which the condition of acute obstruction

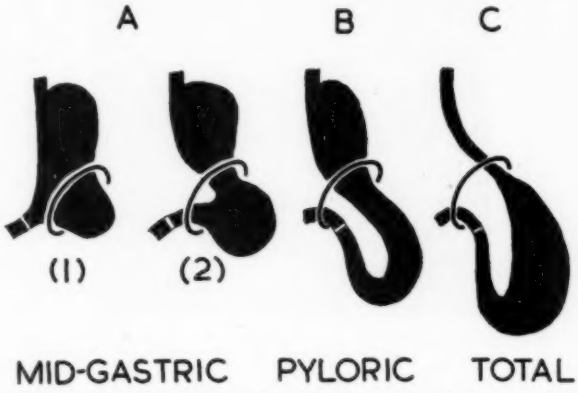

FIG. 803.—Classification of stomach in inguinal hernia and possible method of descent.

has been treated successfully by conservative measures and elective surgery performed after the diagnosis had been made by an opaque meal.

DISCUSSION

Type of Hernia.—Except for the cases described by Brunner (1897) and Novaro (1930), the inguinal herniæ containing stomach have all been very large ones and have frequently been described as the size of a man's head. As with most large herniæ, they had been present for many years. In 23 patients the stomach was in a left inguinal hernia, in 10 in a right, and in 1 patient the side was not recorded; 13 of these patients had bilateral herniæ.

Only in Novaro's case was the stomach the sole content of the sac, and it has usually been accompanied in varying degree by the omentum and large and small intestines. Generally only a portion of the stomach has been found in the sac, and Chambard (1912) classified these herniæ as mid-gastric and pyloric, according to which part of the stomach was in the sac (*Fig.* 803 A, B). He further subdivided the mid-gastric group into those with a diverticulum of the greater curve and those with a complete segment of the stomach, including the lesser curve, in the sac (*Fig.* 803 A 1, 2). However, in Rieder's patient, who was examined with opaque meals over a period of three and a half years, the greater curve was the first portion to go into the hernial sac, followed two years later by the pyloric part of the stomach. *Fig.* 804 is the print of an opaque meal that we had done on a patient with a very large scrotal hernia to determine if it contained the stomach. Although the stomach had not entered the sac, it shows what might be a commencing hernia, developing as a diverticulum of the greater curve. Lewin (1893), Elischer (1923), Rodzevich (1935), and Alexsandrovskiy and

FIG. 804.—Radiograph of a barium meal in a man with a very large left inguinal hernia. Is the greater curve about to descend as a diverticulum into the hernial sac?

Degtyarev (1940) have all described cases in which the complete stomach was in the sac (*Fig.* 803 C).

Mechanism of Descent of Stomach.—Chambard (1912), Sicot (1927), and Novaro (1930) have discussed in detail the mechanism of descent of the stomach, but we think it may be simpler than they suggest, even though a number of factors must be involved to produce such a rare occurrence. It is common experience that the omentum is almost invariably present in large inguinal herniæ and is frequently adherent to the wall of the sac. It seems likely that as the sac increases in size, traction is exerted on the adherent omentum, which in turn exerts traction on the stomach, drawing it down into the hernia. The foregut seems to be fixed most securely at two places, namely the cardia or high lesser curve of the stomach, where the left gastric vessels join the stomach, and where the common bile-duct joins the duodenum. Prolonged downward traction on the foregut causes it to hang like a hammock between these relatively fixed points. Such traction is caused by the great omentum, and it is easy to imagine how the omentum can lead different segments of this hammock into the scrotum.

After demonstrating the presence of the stomach in an inguinal hernia by radiography, Dreessen (1925) at operation found that the stomach was not in the sac, but by pulling on the omentum he was able to draw part of the stomach into the sac. The omentum thus pilots the stomach into the scrotum and the varying types of hernia are produced according to the attachments of the omentum and which part of it descends into the scrotum first. There is a lot to be said for regarding types A to C (*Fig.* 803) as a progression, A being the simplest form and C the most advanced.

Maag (1920) and Dreessen (1925) noted that their patients were small of stature, as was our patient, and this may be another factor in the descent of the stomach into the scrotum.

Cause of Acute Symptoms.—When acute symptoms supervene, it is because the hernial ring causes an obstruction of the stomach. The effect is either to produce a bilocular stomach with a superiorly distended portion, mostly in the abdomen, and inferiorly a smaller distended portion in the hernia, or else a 'lateral pinching' of the greater curve. In the case recorded by Yvan (1830) the stomach contained 5 litres of fluid. Though circulatory changes may be present, nobody has recorded gangrene of the stomach; small intestine, colon, and omentum, all commonly present in the sac, have usually not been constricted. As Chambard (1912) has said, "The extreme gravity of the symptoms is not due to the danger of gangrene of the stomach but to a high stenosis of the digestive tube". This is confirmed by the dramatic improvement that occurred in our patient following simple aspiration of 137 oz. of fluid from the dilated part of the stomach proximal to the constricting ring.

In two patients (Ahrens, 1920; Elischer, 1923) acute symptoms came on soon after taking a large meal and this may well be a precipitating factor in some cases.

Diagnosis and Treatment.—In any very large hernia the possibility of the stomach being one of the contents should be considered. Palpation of the hernia while the patient is drinking might suggest the presence of the stomach and a barium meal would decide the matter. However, in an uncomplicated hernia it is not of great importance to diagnose the presence of the stomach beforehand.

When obstruction occurs with vomiting and distension and tenderness in the hernia, it would appear from the cases published that the hernia is usually soft and the cough impulse present, though the hernia may have become irreducible because of the abdominal distension, as in our patient. It is in such circumstances that it may be of importance to consider the possible presence of the stomach in the hernia, so that the very ill patient may be treated by gastric suction and intravenous therapy rather than operation. If the acute symptoms subside, operation can then be deferred until the patient is in a fit condition for it.

STOMACH IN FEMORAL HERNIA

CASE REPORT

HISTORY.—L. R., a man of 68, had been in hospital since the age of 51 because of mental deficiency with persecutory ideas and hallucinations. When he was admitted to the mental hospital his large hernia was noted. He had no dyspeptic symptoms and the only record of any upset possibly due to his hernia was a short bout of vomiting when he was 61. However, when this occurred his hernia remained reducible and it appears that it only became irreducible about the age of 66. It was learned

FIG. 805.—Photograph of a femoral hernia containing part of the stomach (*see Fig.* 806).

that he worked well in the ward and frequently polished the floors. His hernia did not appear to trouble him.

ON EXAMINATION.—Physical condition reasonable for age. Height 65½ in. (166 cm.). He had a large globular left femoral hernia which was 12 in. (30 cm.) in diameter (*Fig.* 805). Coils of small intestine were readily palpable in the hernia and when he lay down it was only possible to reduce some of the contents. The hernia was resonant to percussion and there was no appreciable alteration in the physical signs after drinking over 20 oz. (570 ml.) of water. His right testis was palpable in the scrotum, but the left could not be defined.

A barium meal, done under considerable difficulty because of the lack of co-operation of the patient and the size of the parts, showed the lower part of the body of the stomach and the pyloric antrum in the hernial sac (*Fig.* 806).

TREATMENT.—Surgical repair of the hernia was not indicated in view of his mental condition, age, and the absence of symptoms.

REVIEW OF CASES ALREADY PUBLISHED

Table II is a summary of the 3 cases already published of stomach in femoral hernia. Cave (1948), in addition to reporting a new case, cited three already published. However, one of these (Ahrens, 1920), with a rather misleading title, was actually a case of stomach in an inguinal hernia and we have recorded it under that heading.

Keller (1885) was the first to record a case of stomach in a femoral hernia. His patient, a spinster of 47, was moribund when brought into hospital and the diagnosis was made at autopsy, when a huge distended bilocular stomach was found. The smaller loculus, 15 cm. in diameter, was in the hernial sac, and the larger proximal one was in the abdominal cavity. According to the patient's landlady, she had had stomach trouble for eighteen years, but in

Table I.—SUMMARY OF PUBLISHED CASES OF STOMACH IN INGUINAL HERNIA

Table I.—SUMMARY OF PUBLISHED CASES OF STOMACH IN INGUINAL HERNIA

No.	Author and Date	Sex	Age	Side	Symptoms and Signs	Size	How Diagnosed	Gastric Content	End-result	Remarks
1	Lallement, 1802	M.	32–64	R.	Pain. Vomiting. Hernia enlarged after eating	Enormous	Autopsy	Pylorus	Death (hernia)	Diagnosis probably suspected during life
2	Yvan, 1830	M.	Not recorded		Vomiting	Enormous	Autopsy	Pylorus	Death (hernia)	5 l. of fluid in stomach
3	Fabre, 1832 Lebert, 1838	M.	73 78	R.	Symptom-free	To knees	Clinically. Confirmed at autopsy	Pylorus	Death (other causes)	Fluid thrill in hernia while drinking
4	Fogt, 1884	M.	60	L.	Vomiting	Man's head	Autopsy	Pylorus	Death (hernia)	—
5	Schmidt, 1885	M.	65–70	L.	Hæmatemesis. Hernia tender	Man's head	Autopsy	Middle two-fourths	Death (hernia)	Diagnosis suspected
6	Chiari, 1888	M.	74	R.	None	To knees	Autopsy	Pylorus. Duodenum	Death (other causes)	—
7	Lewin, 1893	M.	53	L.	Pain. Vomiting. Hernia enlarged and irreducible	To knees	Autopsy	Whole stomach	Death (hernia)	All digestive tube except rectum and duodenum in sac
8	Chevereau, 1894	M.	77	L.	Pain. Vomiting	Adult head	Autopsy	Pyloric region	Death (hernia)	—
9	Souligoux, 1896	M.	?	L.	Not recorded	Enormous	Autopsy	Pylorus	Death (cause not recorded)	Direct and indirect sacs. Stomach in direct sac
10	Brunner, 1897	M.	28	L.	Pain. Hernia enlarged and irreducible	Medium	Emergency operation	Greater curve	Recovery	First patient operated upon
11	Hilgenreiner, 1910	F.	52	L.	Pain. Vomiting	Man's head	Emergency operation	Pyloric third	Death (hernia)	Intra-abdominal volvulus of small intestine
12	Chambard, 1912	M.	62	L.	Pain. Vomiting. Hernia irreducible	Adult head	Emergency operation	Greater curve	Death (hernia)	Died 7th post-operative day
13	Rieder, 1915	M.	65	L.	Hæmatemesis. Melæna	To lower third thigh	X rays	First, greater curve; later, pylorus	Not recorded	First X-ray observation. Gradual descent of stomach into hernia
14	Maag, 1920	M.	81	L.	Symptom-free	Sac 12 cm. wide	Autopsy	Pylorus	Death (other causes)	Small in stature
15	Ahrens, 1920	M.	40	R.	Pain. Hernia enlarged	Man's head	Emergency operation	Not recorded	Recovery	Operation in 1900
16	Stokes, 1922	M.	42	R.	Vomiting. Hernia irreducible	Distended bladder	Emergency operation	Major portion stomach	Recovery	Indigestion prior to acute symptoms
17	Elischer, 1923	M.	70	L.	Hernia irreducible	Man's hat	Emergency operation	Total stomach	Recovery	—
18	Elischer, 1923	M.	53	L.	Nausea. Hernia irreducible	Man's hat	Emergency operation	Total stomach	Recovery	15 l. gas and fluid removed from stomach
19	de Vernejoul and de Luna, 1925	F.	57	L.	Not recorded	Very large	X rays	Not recorded	Recovery	First patient treated by elective surgery
20	Dreessen, 1925	M.	62	L.	Pain. Vomiting. Hernia enlarged after drinking. Splashing sound	Child's head	X rays	Pylorus	Recovery after operation	At operation stomach not in sac but drawn into it by pulling on omentum
21	Sicot, 1927	M.	59	L.	Mild dyspepsia. Sudden pain and vomiting. Hernia enlarged	Fairly big	Emergency operation	Pylorus	Recovery	Perforated gastric ulcer in herniated part of stomach

Table I.—SUMMARY OF PUBLISHED CASES OF STOMACH IN INGUINAL HERNIA, *continued*

No.	Author and Date	Sex	Age	Side	Symptoms and Signs	Size	How Diagnosed	Gastric Content	End-result	Remarks
22	Lipkin, 1928	M.	60	L.	Strangulation. Hernia smaller after vomiting	Man's head	Emergency operation	Middle greater curve	Death (hernia)	—
23	Siegmund, 1929	M.	?	R.	Not recorded	Very large	Autopsy	Pylorus	Death (cause not recorded)	—
24	Novaro, 1930	M.	55	R.	Pain. Vomiting. Hernia irreducible	Orange	Emergency operation	Most of body of stomach	Recovery	Stomach the only content of hernial sac
25	Rodzevich, 1935	M.	54	L.	Pain. Vomiting. Hernia enlarged	To knee	Emergency operation	Complete stomach	Death (hernia)	—
26	Herrmann, 1937	M.	80	L.	Occasional vomiting	Man's hat	X rays	Pylorus	Not recorded	—
27	Lust, 1937	M.	62	L.	Symptom-free	To mid-thigh	X rays	Two-thirds stomach	Not recorded	—
28	Lemaitre, 1937	M.	51	L.	Dyspepsia on standing	Large	X rays	Middle of stomach with lesser curve	Not recorded	Height 1·64 m.
29	Oakley, 1937	M.	81	R.	Pain in hernia and abdomen	To knees	Emergency operation	Pylorus	Death (hernia)	Perforated gastric ulcer in sac; also gangrenous intestine
30	Alexsandrovskiy and Degtyarev, 1940	M.	73	L.	Strangulation	Adult head	Emergency operation	Complete stomach	Death (hernia)	—
31	Jorge, Feldman, and Iturrioz, 1943	M.	66	R.	Symptom-free	Fœtal head	X rays	Middle of stomach with lesser curve	Death (other causes)	Stomach in abdomen at autopsy
32	Hartley, 1945	M.	67	L.	Dyspepsia	Large	X rays	Middle greater curve	Not recorded	—
33	Lewis and Ross, 1950	M.	69	R.	Occasional vomiting	Football	X rays	Pylorus. Duodenal bulb	Recovery	Had elective surgery
34	Anger, 1952	M.	74	L.	Vague stomach trouble	Enormous	X rays	Pylorus. Duodenal bulb	Not recorded	—

Table II.—SUMMARY OF PUBLISHED CASES OF STOMACH IN FEMORAL HERNIA

No.	Author and Date	Sex	Age	Side	Symptoms and Signs	Size	How Diagnosed	Gastric Content	End-result	Remarks
1	Keller, 1885	F.	47	L.	Pain. Vomiting for 3 days	Man's head	Autopsy	Pyloric region	Death (hernia)	Tall in stature. 5 l. fluid in stomach
2	Spiegel, 1920	F.	55	L.	Strangulation	Child's head	Emergency operation	Whole stomach	Recovery	Patient only 136 cm. in height. Xiphoid to symphysis 18 cm.
3	Cave, 1948	F.	56	L.	Dyspepsia	Large	X rays	First, greater curve, then pyloric antrum, duodenum, and pars media	Not recorded	Short stature

spite of this her favourite food was roast potatoes and it was nothing unusual for her to consume 2-3 lb. of them at a single meal. One of these large meals seems to have precipitated the acute obstructive episode that led to her death. Although her hernia was the size of a man's head, apparently nobody knew of its existence.

Spiegel (1920) is the only person to have recorded a case submitted to surgery, and his patient recovered from the emergency operation done for suspected strangulated femoral hernia. Cave (1948) made the diagnosis in his patient with a barium meal, and was the first to record a case diagnosed in this way.

FIG. 806.—Radiograph of barium meal showing part of the stomach in a left femoral hernia (patient shown in *Fig.* 805).

Comment.—Our case of stomach in a femoral hernia is the first to be recorded in a male patient and is only the fourth to be published. It is, therefore, an extremely rare condition.

We have already discussed the type of hernia, mechanism of stomach descent, the cause of acute symptoms, and the diagnosis and treatment in the case of stomach in inguinal hernia, and the same conclusions would appear to be applicable to the much rarer occurrence of stomach in femoral hernia. We would only add that in all 4 cases it was into a left-sided femoral hernia that the stomach descended.

SUMMARY

It is very unusual for the stomach to be a content of either an inguinal or a femoral hernia.

Stomach in Inguinal Hernia.—

1. Over a period of one hundred and fifty years, 34 cases of stomach in inguinal hernia have been reported in the literature, and only 5 of these have been in the English language. A summary is given of the published cases.

2. A further case is described, which appears to be the first recorded in which acute obstructive symptoms were treated successfully by gastric suction and intravenous therapy and elective surgery performed after the diagnosis had been made by a barium meal.

3. Any very large inguinal hernia may contain the stomach, which has been found more often on the left side. The pyloric end is the part most commonly found in the sac.

4. The condition has been diagnosed at autopsy by an opaque meal or at emergency operation when acute obstructive symptoms have simulated a strangulated inguinal hernia.

5. The omentum appears to be the main factor in guiding the stomach into the hernia. Smallness of stature may sometimes be a subsidiary factor.

6. The principal complication is constriction of the stomach by the hernial ring causing obstruction and distension of the stomach, as shown by vomiting and abdominal distension. The physical signs resemble strangulated hernia, but seem usually to be sufficiently atypical to suggest the correct diagnosis and a trial of gastric suction and intravenous therapy rather than emergency operation.

Stomach in Femoral Hernia.—

1. The stomach is so rarely found in a femoral hernia that only 3 cases have been reported in the literature. A summary is given of these cases.

2. A further case is described, which is the first to be recorded in a male patient.

3. In all 4 cases the stomach has been in a left-sided femoral hernia.

We wish to thank Drs. F. L. Rayner and W. Stachurko for X-ray examinations, and Drs. J. Bruegel, P. D. Campbell Jackson, B. W. Jaslowitz, and Mr. S. Marenko for translating articles.

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CONGENITAL ARTERIOVENOUS FISTULA IN THE EXTREMITIES

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" To determine the cause of a disease often leads to the remedy."—*Celsus, Lib.* 1, Prefatio.

IN the past the only certain cure for giant limb has been amputation. In one such case treated by the writer there was found to be a congenital arteriovenous fistula (A.V.F.) in the tibia. Bony A.V.F. are reputedly very rare, but it may be more common than is usually believed and they may be responsible for the failure of conservative operations for giant limb. The many aspects of A.V.F. have been described by Reinhoff (1924), Reid (1925), Pemberton and Saint (1928), Lewis (1930), Holman (1937), and others, but one fact stands out clearly—ligation of vessels is often followed by amputation. Few cases have been cured, and then only when the fistula was in the peripheral vessels and small enough for complete excision.

FIG. 807.—The patient at 29 years of age. The left lower limb is 3½ in. longer than the right, 2 in. in the tibia and 1½ in. in the femur.

FIG. 808.—The ' hot ' ulcer in the lower third of the leg. There was a continuous oozing of bright red venous blood. Note the cutaneous hæmangioma.

I have found only 6 cases of congenital A.V.F. in bone in the literature. In 1867 Gross (quoted by Lewis) reported one in the leg that required amputation. The vessels were injected with tallow and magnesia and " the injection mass escaped not only from the superficial and deep veins, but escaped from the medullary canal of the tibia and fibula ".

In 1925 Reid reported an extensive A.V.F. in the tibia. In 1930 Lewis described an A.V.F. in a forearm. Radiography showed that the radius was honeycombed by an arteriovenous communication in it. He did not suggest that the intra-osseous fistula should be excised, although he does stress that this is one of the difficulties. He reviewed the cases that were amputated and stated that multiplicity of the fistulæ in the peripheral vessels was the main difficulty, but it is evident that these are difficult or impossible to demonstrate, either at operation or when the limb is dissected after amputation. It seems to me not improbable that in these cases there was an unsuspected A.V.F. in the bones, although this cannot be proved because the bones were not examined. Clay and Blalock (1950) reported two cases of congenital A.V.F. in the mandible. One was cured by partial excision of the mandible, and the other was unsuccessfully treated by ligation of the feeding vessels. In 1953 I made a preliminary report of the case described in detail below. This was a twenty-nine-years-old woman who had all the classical signs and symptoms of a congenital A.V.F. in the lower extremity. The fistula was not in the peripheral vessels but in the tibia, as was shown after amputation.

FIG. 809.—Trophic ulcer in the sole of the foot. This is an important distinguishing feature between ulcers from congenital arteriovenous fistula and varicose veins.

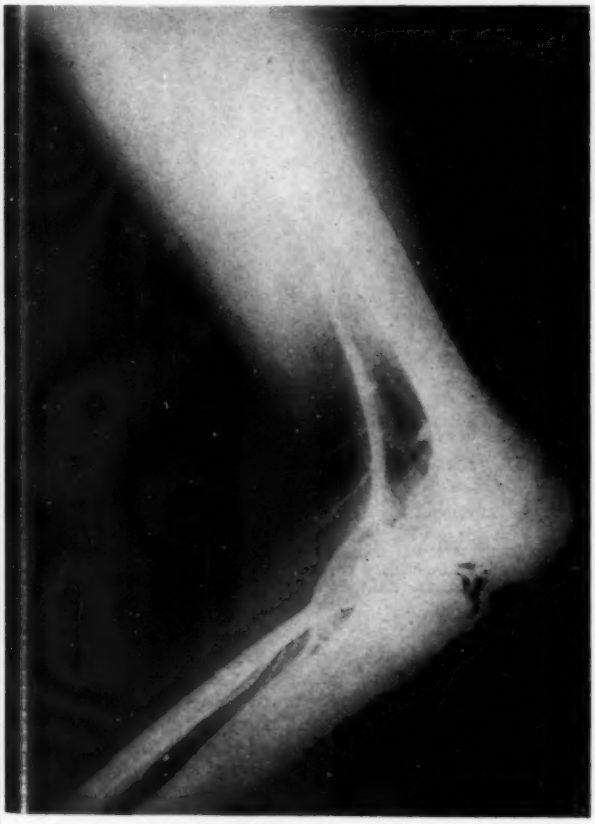

FIG. 810.—Arteriogram taken in 1939. The dye rapidly collects in the subcutaneous veins at the knee, which was the site of a bruit.

FIG. 811.—Arteriogram taken in 1951. This shows a normal arterial tree. The large vessels arising from the bifurcation of the popliteal artery is the feeding vessel to the intra-osseous fistula.

CASE REPORT

M. H., female, aged 29 years at the time of amputation in 1952. At birth the left lower limb was blue and bigger than the right. The discrepancy was 3½ in. when growth had ceased, 2 in. in the tibia and 1½ in. in the femur (*Fig.* 807). This was confirmed by marker radiographs. From twelve onwards trophic ulcers appeared, first in the toes and then in the sole of the foot and leg (*Figs.* 808, 809). Uncontrollable oozing of bright red venous blood from the ulcers made amputation necessary. At 13 a thrill appeared in the knee, leg, and foot. She

was admitted in 1938 for skin-grafting, and Mr. Murray Falconer diagnosed congenital A.V.F. in the leg. Arteriography suggested an A.V.F. in the popliteal fossa (*Fig.* 810). Later further arteriography, and also venography and medullary bone punctures, were done in attempts to localize the fistula.

AT OPERATION (Mr. Renfrew White, 1939).—A large branch of the popliteal artery was found that " seemed to dive deeply and may have entered the tibia ", and was ligated. After this the patient said that the thrill in her leg was less. During the next ten years she remained well, except for the ulcers breaking down from time to time. She married and had three children. In 1951 the limb was indurated, hot, and the subcutaneous veins were pulsatile, tortuous, and enormously dilated. The bruit was still present. No abnormality was detected in an arteriogram at this time, although it was suspected that the fistula was in the bone, but review of the films subsequently showed a large artery entering the tibia (*Fig.* 811). Oxygen saturation in the venous blood from the left foot was 81 per cent and from the right 41 per cent. Large pulsatile veins were ligated by Mr. James Jenkins and myself, but the only improvement was a disappearance of the bruit. Arterial bleeding from the ulcers continued. The knee was disarticulated because of the uncertainty of stopping the bleeding if the fistula had proved to be in the tibia. The stump healed well and a satisfactory limb was fitted. At present (1953 the distal third of the stump is hot and it is probable that there is an A.V.F. in the femur. The heart was never enlarged (radiograph 1951) and the pulse and blood-pressure were always normal.

FIG. 812.—The tibia has been stripped to the periosteum. The feeding vessel is seen to arise from near the bifurcation of the popliteal artery. It is the same size as the main vessels. The expanded area of the shaft is due to dilated veins in the periosteum.

FIG. 813.—The tibial shaft split longitudinally. The feeding vessel was traced into the black area in the middle third of the medullary cavity. In the injected specimen this area was bright red. Note the calcified crescentic lower margin.

Examination of the Amputated Limb.—The main artery of the leg was injected with red lead and gelatin. The peripheral vessels of the leg and foot were traced throughout their course, but no A.V.F. was found. The venæ comites were empty, but some of the peripheral veins had filled. An artery, about 2 in. long, arose from near the bifurcation of the popliteal, and entered an expanded portion of the middle third of the tibia (*Fig.* 812). Dr. Rodda pointed out that this vessel, which was quite distinct from the nutrient artery, had nearly the same calibre as the popliteal artery. It had no venæ comites and was histologically normal. The apparent expansion of the tibia was due to dilated periosteal veins, one of which had made a deep groove in the bone. The blood had exit by these veins, particularly towards the proximal extremity of the shaft. The feeding vessel was traced by

splitting the tibia longitudinally. It entered a densely injected area in the middle third of the shaft that had completely replaced the lamellar bone. The lower edge was bounded by a calcified crescentic margin (*Fig.* 813). Histological examination showed blood spaces lined by a single layer of endothelium (*Fig.* 814).

FIG. 814.—Photomicrograph of the fistulous area shown in *Fig.* 813. The normal lamellæ were replaced by blood-sinuses lined by a single layer of endothelium. (H. and E.) (× 135.)

DISCUSSION

The difficulties in treating congenital A.V.F. in the extremities are due either to multiplicity of the fistulæ in the soft tissues or to an A.V.F. in bone, or to a combination of both. I think that the possibility of an intra-osseous site has not been sufficiently studied in the surgical approach to this disease. If a bony fistula is present and not excised then recurrence is inevitable. Ligation of the feeding vessels is not enough because new arteries enlarge and replace them. To cure this disease I visualize an operation done in stages. First, ligation of the feeding vessels and any fistula found in the peripheral vessels. Secondly, localization and excision of the intra-osseous fistula, and packing of the cavity with stored cancellous bone to stop the bleeding. Diaphysectomy may be possible in the fibula and forearm bones. And, thirdly, restoration of the shaft by bone-grafting if necessary. I have not yet had the opportunity to do this.

SUMMARY AND CONCLUSIONS

1. Congenital A.V.F. in the extremities is an intractable disease.

2. It is suggested that a search for the fistula must include the bone.

3. An operation that might possibly cure the condition has been described.

I am obliged to Dr. Charles Begg for his painstaking radiological examination of the blood-vessels; and to Dr. Rodda, who injected the limb and helped me with the dissection; to Miss Marshall for the prints and the photomicrograph, and to Miss L. North for secretarial aid.

I am obliged to Professor Sir Gordon Bell and Professor Woodruff for their help and criticism with this study.

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SHORT NOTES OF RARE OR OBSCURE CASES

A CASE OF FATAL ASPHYXIA DUE TO INFECTION OF A VENTRICULAR LARYNGOCELE

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Dilatation of the laryngeal ventricle, for which the term ' laryngocele ' was introduced by Virchow (1867), appears to be a very rare condition. Reviewing the literature and recording a further case, Taylor (1944) was able to trace reports of 35 cases, while Freeman (1952), reporting 3 cases, found another 3 in the literature between 1944 and 1952. I have not been able to find any report of a case with fatal complications.

Fig. 815.—The specimen removed at autopsy, showing the larynx displaced to the right by a large mass lying between the larynx and the hyoid bone.

Fig. 816.—The specimen with the trachea opened from behind; the tracheotomy opening is seen in front of the rod separating the tracheal walls.

Three types of laryngocele are said to occur (Lindsay, 1940), viz., internal, superior external, and mixed, the last being a combination of both former types. The internal laryngocele, lying within the larynx, is the most commonly described; the superior external laryngocele, which bulges through the thyrohyoid membrane and presents a swelling in the neck, and the mixed type are both rare. The aetiology of the condition does not seem to have been established, factors which have been incriminated in various cases include chronic cough, straining at stool or micturition, childbirth, blowing wind instruments, and lifting heavy weights, while in some cases congenital abnormalities of the laryngeal ventricle have been suggested and in others disease of the laryngeal walls (e.g., tuberculosis) has been clearly responsible. Once herniation through the thyrohyoid membrane has occurred, however, any expiratory effort against a closed glottis will increase the size of the sac.

CASE REPORT

A coal miner, aged 42 years, was brought to Salford Royal Hospital at 5 a.m. on March 27, 1953, *in extremis*. Two years previously he had been investigated elsewhere

for 'laryngitis', no abnormality being found and no treatment prescribed. Twenty-four hours previously a swelling appeared in the left side of his neck and he lost his voice. One hour before his arrival in hospital he awoke with respiratory distress that progressed rapidly. On admission to hospital he was unconscious, sweating, and deeply cyanosed, with a slow full pulse.

There was almost complete laryngeal obstruction, with almost no air entry to the lungs despite violent respiratory efforts which were beginning to fail. There was a firm, mobile swelling, 3 cm. in diameter, to the left of the thyroid cartilage. Direct laryngoscopy, performed at once, revealed the glottis so far displaced to the right that the cords could not be visualized and intubation was clearly impossible. Laryngotomy was therefore performed immediately and a tube inserted through the cricothyroid membrane. A free airway was

FIG. 817.—The same specimen after opening the laryngocele from in front, showing the astonishing restoration of normal anatomy.

obtained, but both respiration and circulation ceased at this moment and artificial respiration with oxygen, intravenous nikethamide, and cardiac puncture failed to produce any response.

POST-MORTEM EXAMINATION.—No abnormality was found except in the respiratory system. There were strong pleural adhesions on the left side and both lungs showed congestion and œdema. There was a cystic swelling about 2·5 cm. diameter on the left side of the larynx at about the level of the vocal cords. This swelling had pushed the epiglottis over to the right and downwards, and the vocal cords were swollen and in close apposition (*Figs.* 815, 816). The swelling was found to be a diverticulum containing air under pressure and a small amount of pus. When the diverticulum was opened air escaped audibly and the anatomy of the larynx was instantly restored to normal, save only for slight œdema of the vocal cords (*Fig.* 817). Dissection showed the diverticulum to be a laryngocele bulging on the one side through the thyrohyoid membrane and on the other pressing into the larynx, mainly into the upper chamber, and also compressing the vocal cords. The cavity was lined by columnar epithelium; there was a little round-cell infiltration of subepithelial tissue, and there were a few collections of polymorphs on the mucosal surface. Communication with the larynx could not be demonstrated.

DISCUSSION

This was a case of mild infection in a superior external laryngocele. It seems that the infection caused œdema of the walls of the laryngocele, narrowing the opening into the larynx and making it valvular in effect. Once this condition was established the patient's increased expiratory efforts inflated the sac further and the vicious circle so produced terminated in asphyxia.

Aspiration of the swelling was considered when the patient was first seen, but was rejected since the diagnosis appeared to be cervical abscess with respiratory obstruction due as much to inflammatory œdema of the glottis as to direct pressure. It was felt that valuable time possibly wasted on attempted aspiration would be better employed on immediate tracheotomy. Possibly aspiration might have been life-saving in this case, though in view of the very short time interval before death I think there was almost certainly irretrievable cerebral damage already present.

SUMMARY

The literature on laryngoceles is briefly reviewed and a case of fatal asphyxia due to an infected laryngocele is reported. No aetiological factors could be incriminated.

I wish to thank Dr. Robert Herring, who performed the pathological examinations and produced the photographs, for permission to publish this case.

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INTRA-OSSEOUS LIPOMA OF RIB

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LIPOMATA which present as easily recognized intra-osseous tumours with expansion of the cortical outline of the affected bone are extremely rare. A survey of the literature has revealed 2 case reports in which the medullary origin of the lesion appears certain (Wehrsig, 1910; Dickson, Ayres, Mason, and Miller, 1951) and a further case in which there is some doubt as to whether the origin may have been periosteal (Caruolo and Dahlin, 1953), whilst Cornil and Ranvier (1882) stated that they had seen

FIG. 818.—Radiograph showing the sharply defined tumour with a trabecular pattern.

an example but give little descriptive detail. In the case forming the subject of this paper it seems at least highly probable that the tumour arose within the substance of a rib.

CASE REPORT

The patient, a man aged 40, was admitted to hospital on account of post-traumatic headaches which had been present ever since a fracture of the skull in childhood, but had recently increased in severity. A routine radiograph of the chest revealed a symptomless lesion of the posterior end of the left first rib (*Fig.* 818). This was in the form of a discrete rounded swelling about 5 cm. diameter, most of which was covered by a thin layer of cortical bone, while several widely spaced trabeculæ could be seen running through it. The possibility of polyostotic fibrous dysplasia was considered, but no other similar lesions were found and since a neoplasm, probably an osteoclastoma, became the provisional diagnosis, it was decided to excise the mass.

AT OPERATION.—On July 6, 1953, Mr. C. Grimshaw made a thoracoplasty incision and confirmed that there was a tumour arising from the posterior half of the left first rib. There was smooth indentation of the apex of the lung, and when the pleura was opened the lung fell away freely. The outline of the lesion in other directions was less clearly defined than was thought from the radiograph, and there appeared to be extension through the soft tissues around the second rib and involvement of the transverse processes and bodies of the adjacent vertebræ. The posterior end of the second rib was excised, followed by the first rib with the greater part of the tumour. Such of the remainder of the lesion as was accessible was removed, but excision was necessarily incomplete owing to the proximity of vital structures. Post-operative recovery was uneventful and the patient has so far remained well.

THE SPECIMEN (RISH 4085/53).—This included a portion of the first rib 9 cm. in length bearing a tumour 5 × 3 × 2 cm. near one end (*Fig.* 819); a portion of the

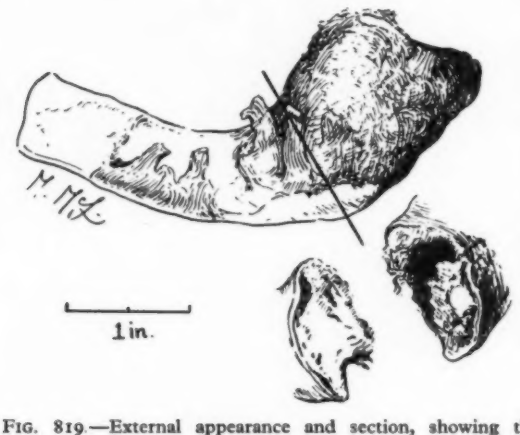

FIG. 819.—External appearance and section, showing the surrounding cortex.

second rib 12 cm. long; and several small pieces of fatty tissue and bone chippings from the affected vertebræ. The main tumour mass was of pale yellow fatty appearance and it occupied the medullary cavity of the rib, being covered over the greater part of its surface by a narrow expanded layer of cortical bone and intersected by widely separated bony trabeculæ. The general form was almost spherical, with conical projections along the medullary cavity at either end, and the limits of extension along the length of the rib were sharply defined. There were areas on the upper and posteromedial aspect of the tumour of more ragged appearance, where fatty tissue had been cut across during removal and no bony capsular layer was present. Here small portions of muscle were adherent.

Microscopically the tumour consisted of mature adipose tissue with sheets of large polyhedral fat cells having a single peripheral nucleus (*Fig.* 820). No embryonic cells and no pleomorphism or other feature suggestive of malignancy was seen in the sections examined. Groups of fat cells were separated by narrow vascular fibrous septa and more widely spaced bony trabeculæ. At the margin not covered by cortical bone there was extension between bundles of striated muscle-fibres.

DISCUSSION

There appears to be no adequate reason why a lipoma should not arise in any site where adipose tissue is present, and this is plentiful in the medullary cavity of bones. Dickson and others (1951) state

that intra-osseous lipomata of flat bones, particularly vertebræ, are not infrequent, although in long bones they are rare. Until an intra-osseous lipoma causes expansion of the bone wherein it lies, it is difficult to be certain that one is indeed dealing with a tumour and not simply an accumulation of fat. Thus collections of pure fatty tissue described

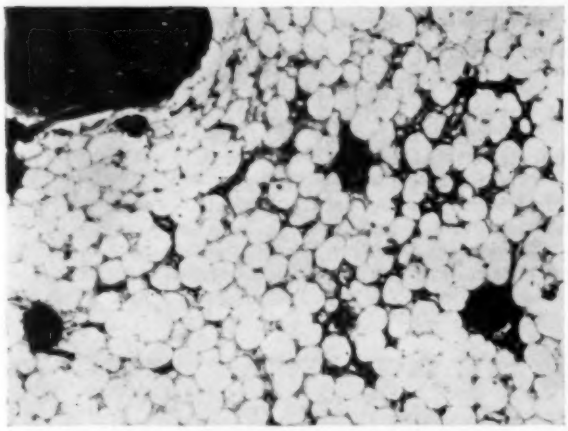

FIG. 820.—The lipoma, showing vascularity and part of a bony trabecula. (× 90.)

in the vertebral bodies may be lipomata, but of this there is no proof (Herzog, 1944).

Wehrsig (1910) described a medullary lipoma of the fibula in a girl aged 5½ which produced a large slightly lobulated swelling of the upper third of the bone entirely covered by a cortex about equal in thickness to that of the normal fibula. This was resected, with a satisfactory post-operative course. Dickson and others (1951) reported the case of a man aged 30 with a history of pain in the ankle after exercise for fourteen years and slight swelling for two years. A radiograph showed a large oval area of rarefaction 8 × 5 cm. in the distal end of the tibia. There were coarse trabeculations crossing part of the translucent area and slight expansion of the shaft. Exploration revealed an apparently normal periosteum and cortex over the anterior aspect of the swelling. Fatty material was removed through a window in the cortex and the cavity was packed with chippings of cancellous iliac bone. This resulted in the disappearance of symptoms and there was subsequent radiological evidence of progressive filling in of the defect. Microscopically the appearances were typical of a lipoma.

These two are the only detailed case reports of undoubted medullary lipomata of the long bones which have been found. In addition, Cornil and Ranvier (1882) observed that they had seen a large lipoma which developed in the body of the tibia, and in which the lobules of adipose tissue, instead of being bounded by fibrous septa, were separated by osseous trabeculæ. Caruolo and Dahlin (1953) described a subperiosteal lipoma of the upper end of the ulna, part of which could be lifted from its bed while part lay within the bone and had to be removed by chisel. They gained the impression that the tumour arose within the bone but thought it equally conceivable that the site of origin was the under-surface of the periosteum, with subsequent erosion of bone owing to the firm attachment of the periosteum in this region. Among a series of 460 lipomata examined by Geschickter (1934), 2 were said to be in bone, but no further details are given.

A few other lipomata attached to the periosteum have been reported, but these have produced no more than slight cortical deformity and have not involved the medullary cavity. Bartlett (1930) described a periosteal fibrolipoma grooved by but not causing deformity of the humerus of a boy aged 7, and a second similar tumour attached to the periosteum of the tibia of a boy aged 6. In this case there was bowing of the tibia, but no erosion or thinning of the cortex. Geschickter and Copeland (1949) saw a girl aged 15 with a periosteal lipoma which had caused erosion and roughening of the medial condyle of the femur.

Liposarcomata of bone have been reported with rather greater frequency than their benign counterparts (Duffy and Stewart, 1938), although there have been criticisms of the validity of the histological diagnosis in some cases (Barnard, 1931) and suggestions that some tumours alleged to be primary may have been metastatic. This type of criticism almost invariably arises when tumours are reported in uncommon situations, but while lipomata only rarely undergo malignant change, their occurrence within bone must be regarded as collateral support for the existence of primary intra-osseous liposarcomata.

The possibility of a lipoma is worth consideration when an isolated bony swelling showing radiological evidence of rarefaction and intersection by widely spaced trabeculæ is seen, in that resection if complete will be curative while radiotherapy will almost certainly have little effect.

SUMMARY

A case is reported of lipoma of the rib in a man aged 40. A brief review is given of the few other intra-osseous lipomata described in the literature.

I am indebted to Mr. G. C. Laurie Pile for permission to publish this case.

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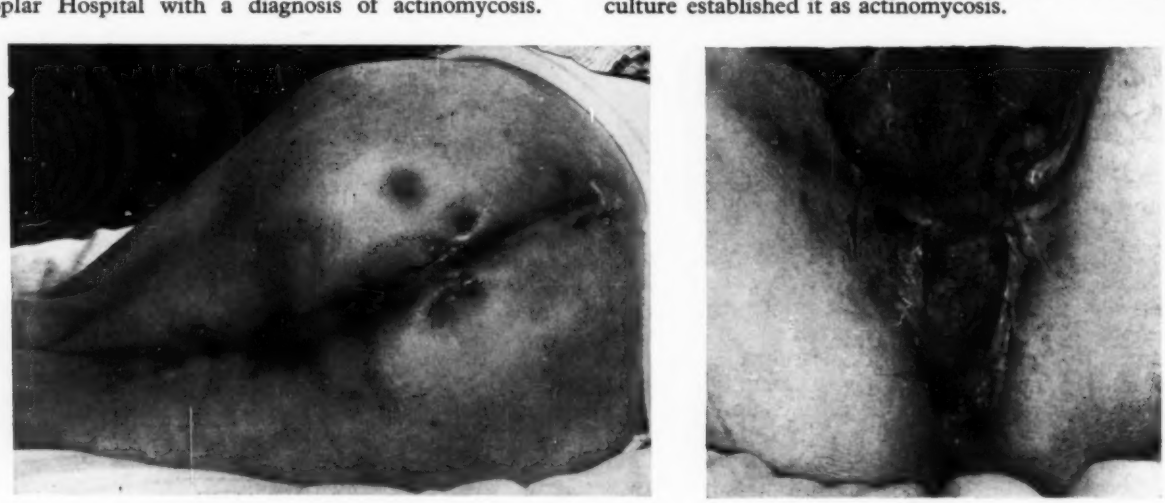
PERIANAL ACTINOMYCOSIS COMPLICATING PILONIDAL SINUS

By A. R. ANSCOMBE and JOHN HOFMEYR, London

Infection of a pilonidal sinus by actinomycosis appears to be a rare condition. One case has been reported (Reichman, 1947) in the American literature, and the following case appears to be the first in this country and the second case recorded. Buie (1944) lists sacral actinomycosis in the differential diagnosis of pilonidal sinus. Cope (1951) records 62 cases of actinomycosis of the spine and adds 3 new cases. In our case, secondary involvement of the coccyx occurred.

CASE REPORT

The patient, a man aged 56, presented in June, 1952, with a 15-year history of recurrent discharge from the sacral region and was admitted under Mr. Alan Perry to Poplar Hospital with a diagnosis of actinomycosis.



A B

Fig. 821.—Actinomycosis of pilonidal sinus. View of sacrococcygeal area showing multiple orifices of fistulæ and sinuses.

Past History.—In early 1937 he complained of an anal discharge and was admitted to the Royal Masonic Hospital. On examination, numerous fistulæ posterior to the anus were seen. Biopsy suggested tuberculous giant cells, but repeated sputa and pus examinations failed to reveal any tubercle bacilli. In a month, the patient was discharged with the area healing.

In 1940 he was admitted to Poplar Hospital where a perforated duodenal ulcer was oversewn. No mention was made in the notes about any ano-coccygeal or sacral pathology.

In 1941 the patient was re-admitted to the Royal Masonic Hospital complaining of a recurrence of the trouble soon after the perforation. Repeated sputum and pus specimens were again negative for tubercle bacilli. Only a mixed flora of contaminents were found. Chest radiograph was normal.

Sir Girling Ball laid open all fistulæ and sinuses. During the next nine months as an in-patient, six further operations were done, and a prolonged course of physiotherapy, including ultra-violet light, was given. On final discharge (in May, 1942) one deep sinus which had been explored and opened three times, was still not healed. No chemotherapy was given, nor, then, any antibiotics.

On Admission (1952).—Generally: Patient was deaf, emphysematous, and hypertensive. Locally: Multiple sinuses of perineum and sacral region, as shown in *Fig.* 821.

Investigations.—

Radiography: Chest, no evident lesion. Pelvis, chronic osteomyelitis of coccyx with sequestrum formation.

During the process of exploring the fistulæ a plug of matted brittle hair and sebaceous material with inspissated pus was extracted from a deep midline sinus.

Frei test and the lymphogranuloma venereum complement-fixation tests were negative.

Congo-red test for amyloidosis—83 per cent of dye still present after one hour. Repeated one month later—result constant.

Bacteriology: After a fortnight's daily search, a Gram-positive, true-branching, beaded streptothrix was found, without any clubbed forms, in the pus. Anaerobic culture established it as actinomycosis.

Histopathology: Biopsy of one sinus showed "sinus lined by granulation tissue, scanty miliary giant-celled tubercles in subcutis. No actinomyces seen. Tubercles not necessarily diagnostic of tuberculosis." (*Fig.* 821 B).

SUMMARY

A case of pilonidal sinus secondarily infected with actinomycosis is reported, and the literature commented upon briefly.

This is believed to be the first reported British case and only the second one in world English literature.

The coccygeal involvement qualifies this also as a case of actinomycosis of the spine.

We wish to thank Mr. Alan Perry for permission to publish this case, and the staffs of Poplar Hospital, the London Hospital, and the Records Department of the Royal Masonic Hospital for their co-operation.

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HETEROTOPIC GASTRIC MUCOSA OCCURRING IN THE LOWER JEJUNUM

By RAYMOND RAMSAY, M.B.E.

FORMERLY SENIOR SURGICAL REGISTRAR, BRISTOL ROYAL INFIRMARY

THE best known example of epithelial heterotopia of the alimentary tract is that of gastric mucosa and pancreatic tissue occurring in Meckel's diverticulum. Numerous cases are described in the literature, where as a result of acute inflammatory change, ulceration, perforation, bleeding, or intussusception, and the attendant symptoms and physical signs, a diagnosis has been made and operative treatment carried out.

Though examples of epithelial heterotopia elsewhere in the alimentary tract, as Taylor (1927) has shown, are not uncommon, the subject is one of considerable theoretical interest only, since there may be little clinical importance attached to these misplaced tissues. An exception, however, is provided by the following case. Here ectopic gastric mucosa was present as a tumour-like mass in the jejunum, causing attacks of subacute intestinal obstruction. I have found no reference of a similar case in the literature.

CASE REPORT

HISTORY.—Valerie G., aged 9 years, was admitted as an emergency to the Bristol Children's Hospital on Jan. 22, 1953. She complained of abdominal pain.

FIG. 822.—The resected jejunum opened on its antimesenteric border. The rugose tumour is shown.

The pain was central and colicky, was said to " double her up ", and had occurred on the day of admission. During the past two months she had had several similar attacks lasting up to 12 hours, usually associated with vomiting and constipation. On one occasion she was admitted and observed for a day. The attack subsided and she was discharged and arrangements made for her attendance at the out-patient clinic.

ON EXAMINATION.—She was a well-nourished, healthy, co-operative child. The temperature was 99° F. and the pulse 100. Her tongue was moist and clean. The chest was clear clinically. The abdomen moved well on respiration, and there was no visible peristalsis.

A soft but firm, rounded, mobile lump was easily palpated to the right of the umbilicus. It moved on respiration and from time to time there was guarding of the right rectus in its upper half. The rest of the abdomen was quite soft and no other mass or viscus was felt. No succussion splash was elicited. Rectal examination was negative.

A diagnosis of subacute intestinal obstruction was made, probably due to an intussusception. Operation was advised and performed immediately.

AT OPERATION.—General anæsthetic of thiopentone, ethyl chloride, ether, and oxygen was administered and a right paramedian incision made. The mass was easily delivered, and proved to be a loop of lower jejunum, dilated and somewhat hypertrophied. At the maximum convexity a soft ovoid mass (4 cm. × 5 cm.) was palpated within its lumen. The bowel was opened along its antimesenteric border, and a clearly defined rugose tumour was seen (*Figs.* 822, 823). The tumour, together with several centimetres of healthy bowel on either side, was resected, and end-to-end anastomosis performed.

FIG. 823.—A side view of the resected jejunum.

POST-OPERATIVE PERIOD.—She made an uninterrupted recovery, and was discharged home on the fourteenth day.

PATHOLOGIST'S REPORT.—The specimen consisted of 10 cm. of small intestine (circumference 6 cm.) with ribbed mucosa of typical jejunal pattern, interrupted in the middle on the mesenteric border by a rugose tumour (3·8 cm. × 5 cm.) resembling gastric mucosa (*Fig.* 824). Cut surface showed the tumour to be due to thickening of mucous membrane. There was no affection of the submucosa, or infiltration of the deeper structures.

A section confirmed the naked-eye view that this is an area of ectopic gastric mucosa. Its pattern is rather irregular and many of the deeper crypts are dilated and penetrate deep to the muscularis mucosa. Oxyntic cells are present. (*Fig.* 825.)

DISCUSSION

The main symptom was one of colicky abdominal pain. This was due to an interruption of normal peristalsis by the presence of the tumour, and perhaps by an attempt at intussusception, which may have been prevented by the fact that the tumour occupied nearly the whole of the circumference of the bowel. Suffice it is to say that the patient's symptoms were due to the presence of the tumour itself and not to any change in either the ectopic mucosa or the nearby jejunal mucosa. This is in marked distinction to gastric mucosal heterotopia in Meckel's diverticulum, where symptoms are caused by peptic ulceration and its complications. As has

been pointed out by Maddock and Coventry (1941), peptic ulcer may occur at the base of a Meckel's diverticulum and in adjoining ileal mucosa, owing to bathing of the more susceptible intestinal mucosa by peptic juices.

It is tempting to suggest that heterotopic tissues in the small intestine may sometimes cause peptic ulcer, and that this factor may be of importance in considering the aetiology of the so-called primary or simple ulcer of the jejunum and ileum. However, gastric heterotopia was not found in the 47 cases of primary jejunal ulcer which were reported in the literature between 1827 and 1932 and reviewed by Ebeling in 1933; nor was it present in 75 cases reviewed by Berry and Dailey in 1940, when they described an additional case. In the cases described, aetiological factors were absent. There was no evidence of typhoid, dysentery, tuberculosis, regional ileitis, parasitic infection, or syphilis; and there was no history of gastro-enterostomy or trauma. No heterotopic gastric mucosa or pancreas was found in any of the cases. The ulcer generally resembled peptic ulcer and varied from 0·5 cm. to 2 cm. in diameter. Spontaneous perforation was common, in other cases vague dyspeptic symptoms occurred, though not infrequently the symptomatology was more clear-cut, resembling duodenal ulcer. Stenosis causing attacks of subacute intestinal obstruction also occurred, and melæna was a presenting symptom in a minority of cases.

Brown and Pemberton (1936) reported on 10 cases of primary ileal ulcer, and stated that no epithelial heterotopia was present, though they discussed the possibility of its presence as an aetiological factor. However, Taylor reported a case of a baby of 17 months who died as a result of hæmorrhage from an ileal ulcer. The histological examination revealed tall gastric fundic glands in adjoining ileal mucosa.

Muller (1921), Stone (1923), and Brown (1924) discussed the possibility of the presence of ectopic mucosa as a factor in the aetiology of primary simple intestinal ulcer, though Nicholson (1923, a, b) maintained that heterotopic gastric mucosa had not been reported distal to the duodenum, except in relation to Meckel's diverticulum.

Taylor (1927) investigated 130 cases of epithelial heterotopia of the alimentary tract; of these, 81 were congenital, the remainder acquired. Of the 81 congenital cases, 51 were examples of superficial and 30 of deep heterotopia. In the superficial group gastric mucosa occurred in the œsophagus in 6 cases, in the duodenum in 2 cases, in the small intestine and at the umbilicus in 1 case, and there were 5 examples in Meckel's diverticulum. Intestinal mucosa was present in the stomach in 36 cases, and in 3 cases at the umbilicus. Duodenal mucosa was present in the stomach in 1 case. In the deep group, the stomach was the site of submucous glands in 1 case, and in another case of intramural cyst. Adenomyoma occurred in the stomach in 6 cases and in the duodenum in 3 cases. Accessory pancreas occurred in the stomach in 6 cases, in the duodenum in 7 cases, and in 3 cases each in the jejunum and in Meckel's diverticulum.

Taylor's series would appear to confirm the clinical view that superficial epithelial heterotopia occurs most commonly in the œsophagus, in Meckel's diverticulum, and at the umbilicus; and that deep heterotopia in the form of pancreatic rests commonly occurs in the stomach, duodenum, small intestine, and Meckel's diverticulum. There was only 1 case of gastric mucosa occurring in the small intestine, which was the baby with the ileal ulcer previously mentioned.

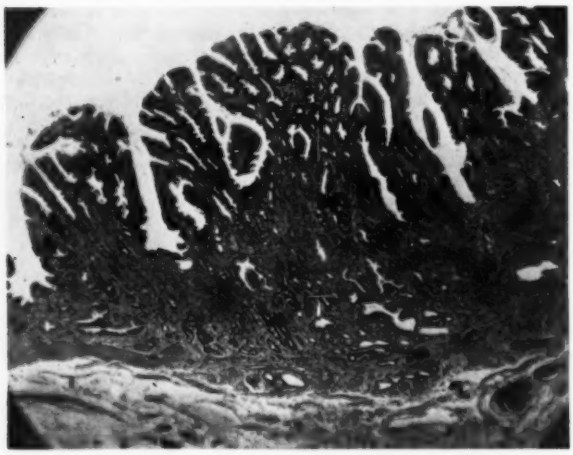

FIG. 824.—Jejunal tumour due to ectopic gastric mucosa (mucosa and submucosa shown). The epitheleium is irregular, and some of the crypts extend deep to the muscularis mucosæ, but otherwise it is typical gastric mucosa. (× 25.)

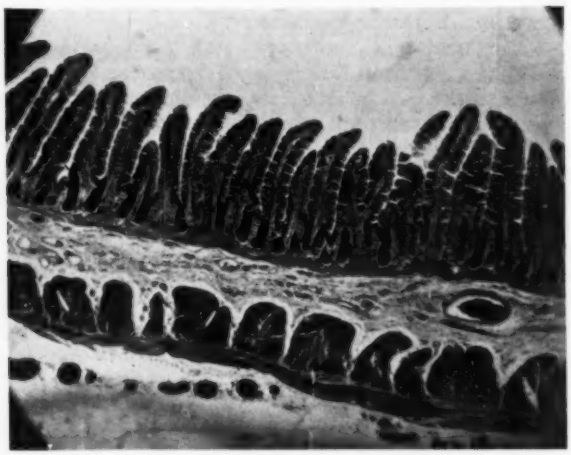

FIG. 825.—Normal jejunal mucosa and muscularis in neighbourhood of tumour. (× 50.)

SUMMARY

A case of ectopic gastric mucosa occurring in the lower jejunum is described. This is a rare site for epithelial heterotopia. The tumour-like mass caused subacute obstruction. The presence of misplaced gastric epithelium is discussed as a factor in the aetiology of primary or simple ulcer of the small intestine.

I wish to record my thanks to Mr. W. A. Jackman, under whose care the case was admitted, and to Dr. O. C. Lloyd for the pathological report and the photomicrographs. I am indebted to Dr. A. L.

Taylor, from whose article I have freely quoted, and to Mr. R. E. Horton for help and advice in preparing this report.

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RENAL TUBERCULOSIS MASQUERADING AS SIMPLE RENAL CYST

BY G. L. GRYSPEERDT AND J. R. THOMPSON
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RENAL tuberculosis is a relatively common condition and the following case is only reported because of the unique manner in which it presented.

CASE REPORT

The patient, a one-legged woman aged 50, was first seen in the Cystoscopy Clinic at St. Thomas's Hospital in July, 1952. She complained of periodic attacks of dysuria and frequency of micturition for the previous six months.

At the age of 5 years a high above-knee amputation had been performed for what was thought to be a tuberculous left knee. In 1936 she was treated for hæmaturia and dysuria at another hospital, and was told at this time that some polyps had been removed from her bladder.

ON EXAMINATION.—The patient was tense and talkative. The left lower limb had been amputated just below the greater trochanter, and there were scars of numerous healed sinuses below the inguinal ligament. The left kidney was easily palpable and the lower pole was felt to be enlarged.

FIG. 826.—Retrograde pyelogram.

Investigations.—

Catheter Specimen of Urine: Acid. Occasional leucocytes. Sterile. Stellar phosphate crystals seen.

Intravenous Pyelography: On the control film the lower pole of the left kidney appeared enlarged. Both kidneys commenced excreting without delay. The right kidney showed no abnormality. The left kidney showed elevation and distortion of the middle and lower groups of calices with stretching and distortion of the renal pelvis from below.

FIG. 827.—Aortography, nephrogram stage.

Cystoscopy: Marked generalized 'cystitis'. Both ureteric catheters could be passed well up the ureters.

Retrograde Pyelography: The pyelogram of the left kidney (*Fig.* 826) confirmed the appearances shown by intravenous pyelography.

Abdominal Aortography: Aortography, by retrograde catheterization through the femoral artery, showed normal vascularization of the right kidney. In the arterial phase the arteries supplying the lower pole of the left kidney were shown to be raised in an arc-shaped displacement and there was a complete absence of any vascular structure in the lower part of the kidney. In the nephrogram stage the left kidney showed a complete absence of contrast filling of the lower pole, but normal vascularization of the upper pole; the margin was sharply delineated, with a smooth curve convex upwards (*Fig.* 827).

A radiological diagnosis of cyst of the lower pole of the kidney was made on the pyelographic and angiographic appearances.

AT OPERATION.—The left kidney was explored on Oct. 6, 1952, through a loin incision with resection of part of the 12th rib.

The lower renal pole was found to be replaced by a collection of four ' cysts '. The remainder of the kidney, the pelvis, and the upper end of the ureter appeared normal. Partial nephrectomy was, therefore, performed.

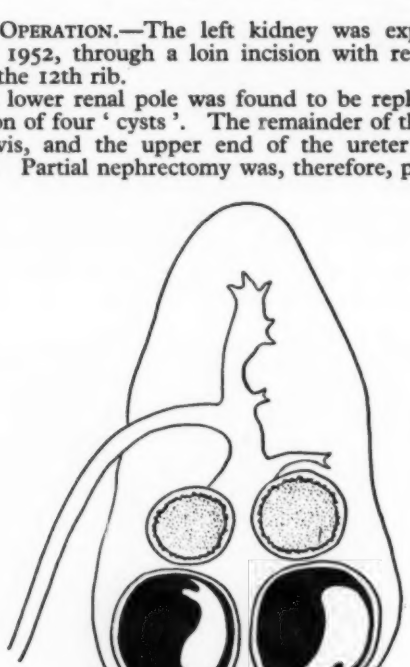

FIG. 828.—Diagram of reconstructed left kidney.

Macroscopical examination of the excised tissue revealed a marked difference between the four ' cysts '. The two proximal cysts were smaller and were lined with granular membranes; they contained serocaseous material. The two distal cysts, on the other hand, were much larger; they were lined with smooth membranes and contained clear serous fluid (*Fig.* 828).

PATHOLOGICAL REPORT.—Caseating tuberculous cyst walls lined with granulation tissue.

DISCUSSION

The clinical signs and pyelographic appearances in this patient strongly suggested the presence of a space-occupying lesion in the lower pole of the left kidney. Renal angiography showed a complete absence of vascular structure in the lower pole of the kidney, with arc-shaped displacement of arteries and a sharply delineated smoothly curved contrast margin in the nephrogram stage—appearances greatly favouring renal cyst rather than neoplasm (Weyde, 1952; Lindgren, 1953).

Exploration of the left kidney revealed a multicystic condition, subsequently proved to be tuberculous in origin.

In retrospect, greater note should have been taken of the patient's symptoms of frequency and dysuria, and a careful search for acid-fast bacilli should have been made. That this was not undertaken was due in large measure to the radiological findings.

SUMMARY

A case of atypical left renal tuberculosis masquerading as a renal cyst is reported. A search of the English literature has failed to reveal a similar reported case.

Our thanks are due to Mr. R. H. O. B. Robinson, under whose care this case was admitted, and to Mr. F. B. Cockett, who kindly performed the aortic catheterization.

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REVIEWS AND NOTICES OF BOOKS

Clinical Orthopædics. Edited by ANTHONY F. DE PALMA, with the assistance of Five Associate Editors and a Board of Six Advisory Editors. Two issues per year. 7 × 10 in. No. 1, Pp. 242 + xiv, with 149 illustrations. No. 2, Pp. 247 + viii, with 232 illustrations. 1953. Philadelphia and London: J. B. Lippincott Co. Each issue, 48s.

THESE volumes devoted to clinical orthopædics constitute a new publication which is sponsored by the Association of Bone and Joint Surgeons of America. Professor De Palma, as editor-in-chief, is assisted by five associate editors and a board of six advisory editors. Therefore, the various chapters in these two volumes may be said to represent the latest orthopædic treatment in America.

The first volume is divided into two main sections; the first is devoted to what is called ' Children's Orthopædics ', surely a curious title; while the second deals with general orthopædics.

The problems of arthritis in children, flared ribs, talipes, Pethes' disease, infantile scurvy, and posture in children are discussed and illustrated in a very practical manner.

Under general orthopædics such conditions as backache and sciatic pain are dealt with, and the question of bone-grafting is discussed, while myositis ossificans is fully described. The illustrations throughout are excellent.

The second volume is devoted to intramedullary nailing and contains an outstanding chapter on " Development of Fracture Surgery during the past 100 years ".

Every aspect of internal fixation of fractures is covered, and one comes to the conclusion that operative intervention in fractures is more common in America than in Britain.

These two volumes cover a wide range and give the reader an excellent idea of the practical extent and standard of orthopædic surgery in America. It is the intention of the editorial board to publish two volumes a year and we look forward with interest to seeing the next volumes.

Klinische Zytologie. Grundriss der Allgemeinen Zytologie und der Zytodiagnostik. By H. J. STREICHER and ST. SANDKÜHLER. 6¾ × 9½ in. Pp. 190, with 57 illustrations. 1953. Stuttgart: Georg Thieme Verlag. DM. 49.80.

As compared with many publications of recent years, this book, entitled *Clinical Cytology*, arouses special interest

because it deals with the subject in a much more comprehensive fashion. Thus the material suggested for examination includes tissues removed at operation, specimens obtained by biopsy, smears from mucous surfaces, and various pathological fluids and secretions. The general part of the book is devoted to a description of the methods and technique employed in making the smear preparations. Then follows a very useful account of the morphological appearances of normal cells and of the changes associated with various pathological processes, including the different types and degrees of neoplastic disease. The special part of the book is concerned with cytological diagnosis and organ cytology. The description of the various cells is given according to the appearances shown by Pappenheim's panoptic stain, but in the chapter on gynæcology by Papanicolaou's method of staining. Other methods of staining are also given in the appendix. The account of the cytology of the various pathological fluids and secretions could in places have been profitably expanded. The illustrations, especially the coloured ones are good, but more of them would have been of advantage. The cytological examination of pathological fluids—sputum, urine, and vaginal secretion—has been increasingly practised in this country during recent years.

Cytology can be employed as an additional means of diagnosis in tissues removed at operation and in biopsy specimens. It can also serve as an alternative to the frozen section in the operating theatre. That this method can be successfully used is shown by the papers published by Dudgeon and his colleagues as long ago as 1927, and more recently by that of Ellis. This is an informative book and should prove a useful guide to those interested in cytological diagnosis.

Thoracic Surgery. By RICHARD H. SWEET, M.D., Associate Clinical Professor of Surgery, Harvard University Medical School. Second Edition. 6½ × 10 in. Pp. 381 + xxv, with 159 illustrations. 1954. Philadelphia and London: W. B. Saunders Co. 50s.

WE are not surprised to see the second edition of this book appear so soon after the first edition was published because the author has given us a book of which the Harvard School may well be proud.

Thoracic surgery, although a comparatively recent branch of surgery, has progressed by leaps and bounds during the last two decades.

This second eidition shows careful revision and some modifications in operative technique which have been introduced since the first edition was published.

The operative technique for such conditions as *pectus excavatum* is the best we have ever seen and the illustrations throughout the whole book are excellent.

The complete range of thoracic surgery has been covered in this volume and we are convinced it will remain as popular as its predecessor.

Surgery for Dental Students. By MICHAEL F. A. WOODRUFF, M.D., M.S. (Melb.), F.R.C.S. (Eng.), Professor of Surgery in the University of Otago, New Zealand; etc. With a Foreword by S. H. WASS, M.S., F.R.C.S., Surgeon, Guy's Hospital. 5⅝ × 8¾ in. Pp. 326 + viii, with 108 illustrations. 1954. Oxford: Blackwell Scientific Publications. 30s.

THIS is an admirable book on general surgery specially written for the dental student. It sets out in an attractive manner the basic principles of surgery which are just as important to the dental student as to his medical counterpart.

Professor Woodruff has made use of his mature clinical background to illustrate in an interesting manner the first principles of surgical diagnosis and treatment. The actual illustrations are excellent and all of them have been carefully chosen.

This book amply covers the examination requirements for the Licentiate in Dental Surgery of the Royal College of Surgeons of England.

Aids to Surgical Diagnosis. By Sir CECIL WAKELEY, Bt., K.B.E., C.B., P.R.C.S. Third edition. 3¾ × 6¼ in. Pp. 252 + viii, with 15 illustrations. 1954. London: Baillière, Tindall & Cox Ltd. 6s. 6d.

THE publishers state that "since its foundation in 1876 the Students Aid Series has proved *indispensable* to each succeeding generation of students . . . many of the volumes are *complete* text-books in themselves".

Few teachers would endorse these views. The series are certainly *useful* to students and many of the volumes *epitomize* text-books. They would be much more useful and ultimately become a better epitome, if they were interleaved with plain pages so that the student could make notes of cases he had seen to illustrate the text, with a précis of the pathology and some line illustrations made by himself. The expense of such an edition might be prohibitive at the present time.

The list of the forty-eight volumes which at present comprise the series indicates the possible breadth of the medical curriculum, and the absurdity of any one being expected to cover it. The publishers, however, have catered widely so as to meet all contingencies.

The author, no less a person than the President of the Royal College of Surgeons of England, gives in the preface a very timely warning to the student, and also to the younger teacher that "aids to diagnosis must always play a secondary part to the clinical examination of the patient".

There are clinicians who refuse to make a diagnosis until all the ancillary services of a large hospital have submitted their reports on the results of the special examinations made in radiology, clinical pathological departments, etc., and then if operative procedure, or autopsy findings prove the final diagnosis to be wrong, a student may be left with the idea that diagnosis is simply a guessing competition, as is also horse racing and results in football pools.

Despite such pious precepts as the foregoing there is a place for this series of 'Aids' if they are properly used. They are small books which fit into the pocket of the male comfortably and easily into the handbag of the female, and, therefore, are available during the odd moments of waiting which are well known to students. They are also eminently suitable as a soporific after an exciting clinical day. The fashion of draping the walls of one's bedroom with clinical aphorisms rather than sports teams and cinema stars might with advantage be revived during these hectic years, e.g., a well-known clinical teacher of the past suggested aphorisms such as "once a bone always a bone", "once a stricture always a stricture". It would not be difficult to collect many more in these days of ultra-specialism and they would also be aids to diagnosis.

French's Index of Differential Diagnosis. Edited by ARTHUR H. DOUTHWAITE, M.D., F.R.C.P., Senior Physician Guy's Hospital, Seventh edition. 6¼ × 10¼ in. Pp. 1058, with 731 illustrations, of which 200 are in colour. 1954. Bristol: John Wright & Sons Ltd. 105s.

THE late Dr. Herbert French first published this book with the title *An Index of Differential Diagnosis of Main Symptoms* in 1912 as companion to the publishers' *Index of Treatment* issued in 1907. He claimed that "it covers the whole ground of medicine, surgery, gynæcology, ophthalmology, dermatology, and neurology", and that it was "an Index in the sense that its articles on the various symptoms are arranged in alphabetical order".

"His aim was to collect all the symptoms and signs that might arise in the course of disease". He wrote

more than half of the original book and produced "a work of reference unique in medical literature". Since its first publication the popularity and usefulness of the book has steadily increased, edition has followed edition, each one thoroughly revised, enlarged, and with additional illustrations. The sixth edition was published in 1945 and Herbert French "resolved to take no active part in any further production". The present edition, which is the seventh, has been carried out by the present senior physician at Guy's Hospital, and one who has worthily carried on the work of his old teacher with the collaboration and help of many contributors, all recognized experts in their subject, and especially does he pay tribute to Sir Adolphe Abrahams.

The title of the book has been changed to indicate the actual contents, but the main characteristics of the original book have, to a great extent, been retained whereby the body of the book still deals with symptoms and the index "gathers these together under the headings of the various diseases in which they occur".

The two parts of the book should be consulted together when trying to correlate some particular symptom with a possible disease, to the diagnosis of which it may be the clue.

It is quite impossible to review such a work as this in any detail. The various articles selected at random and read as critically as one can, are all satisfying, informative, and make the reader consult the index for various cross-references. This is undoubtedly what the present editor had in mind when revising the whole book and there is no doubt he has succeeded.

It should be in every medical practitioner's library whatever his specialty may be. It should also be in every hospital library, so that every resident and visiting member of the staff can consult it. Not only will he find the clue he is seeking but he should be unable to resist continuing to read on and thereby widen his experience and knowledge.

The illustrations, printing, and format are what one expects from the publishers of so many well-known text-books and journals. The price, which may appear large at first sight, will be found to be in no way excessive for what is provided in such an attractive form.

Progress in Clinical Surgery. By various authors. Edited by RODNEY SMITH, M.S., F.R.C.S., Surgeon, St. George's Hospital, London. 6½ × 9½ in. Pp. 414 + x, with 112 illustrations. 1954. London: J. & A. Churchill Ltd. 36s.

THIS is a book of which the surgical profession may well be proud for it is practical, interesting, and informative. It is written by the younger generation of surgeons of this country and they have certainly done themselves credit, for they have selected those surgical subjects in which marked progress has been made during the last decade.

Although this book has been written primarily for the post-graduate student working for the final F.R.C.S., yet we make bold to predict that this volume will be read and re-read with renewed interest by many surgeons who in these days find it difficult to keep up to date.

This book covers recent advances in particular branches of surgery; there are some thirty chapters covering such diverse subjects as: Fluid Replacement in the Surgical Patient; Ulcerative Colitis; Portal Hypertension; Renal Tuberculosis; Progress in Cardiac Surgery; and The Injured Hand.

A stimulating book.

BOOK NOTICES

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest to our readers.*]

Klinische Darmbakteriologie für die ärztliche Praxis. By Prof. Dr. med. et phil. TR. BAUMGÄRTEL, Munich. 6⅝ × 9½ in. Pp. 132 + viii. 1954. Stuttgart: Georg Thieme. DM. 14.40.

Confrontations radio-anatomo-cliniques. Edited by M. CHIRAY, R. A. GUTMANN, and J. SÉNÈQUE. Fasc. v. 10 × 12¾ in. Pp. 70 + iv, with 134 illustrations. 1954. Paris: G. Doin & Cie; Masson & Cie. 1800 fr.

Preservation and Transplantation of Normal Tissues. A Ciba Foundation Symposium. Edited by G. E. W. WOLSTENHOLME, O.B.E., M.A., M.B., B.Ch., and MARGARET P. CAMERON, M.A., A.B.L.S., assisted by JOAN ETHERINGTON. 5½ × 8 in. Pp. 236 + xii, with 55 illustrations, mostly as plates. 1954. London: J. & A. Churchill Ltd. 25s.

Surgical Pathology. By PETER A. HERBUT, M.D., Professor of Pathology, Jefferson Medical College, etc. Second Edition. 6 × 9 in. Pp. 893, with 528 illustrations. 1954. London: Henry Kimpton. 105s.

The Thyroid. A Physiological, Pathological, Clinical, and Surgical Study. By T. LEVITT, M.A., F.R.C.S. (Eng.), F.R.C.S. (Edin.), F.R.C.S.S., Hunterian Professor of the Royal College of Surgeons of England; Lecturer in Surgery to the Fellowship of Post-graduate Medicine at the New End Hospital, Hampstead, London. 7¼ × 9¾ in. Pp. 606 + xxiv, with 502 illustrations, some coloured. 1954. Edinburgh: E. & S. Livingstone Ltd. 105s.

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